A COMPARATIVE STUDY OF RMI 1 AND RMI 2 TO DISCRIMINATE BETWEEN BENIGN AND MALIGNANT OVARIAN TUMORS

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MAY 2019

DECLARATION

DECLARATION

I hereby declare that this dissertation entitled "**A COMAPRATIVE STUDY OF RMI 1 AND RMI 2 TO DISCRIMINATE BETWEEN BENIGN AND MALIGNANT OVARIAN TUMORS"** is a bonafide and genuine research work carried out by me under the guidance of **DR.N.GEETHA M.D (OG)**, Associate Professor, Department of Obstetrics & Gynaecology, Coimbatore Medical College & Hospital, Coimbatore.

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CERTIFICATE

CERTIFICATE

This is to certify that the dissertation entitled "**A COMPARATIVE STUDY OF RMI 1 AND RMI 2 TO DISCRIMINATE BETWEEN BENIGN AND MALIGNANT OVARIAN TUMORS"** is a bonafide and genuine research work carried out by **Dr. S.SUMATHI** in partial fulfilment of the requirement for the degree of Master of Surgery in Department of Obstetrics & Gynaecology.

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Dissertation Topic	:	A Comparitive study of Risk of malignancy index 1 & Risk of malignancy index 2 to discriminate between benign and malignant ovarian tumours.

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted and you are permitted to proceed with the above Study.

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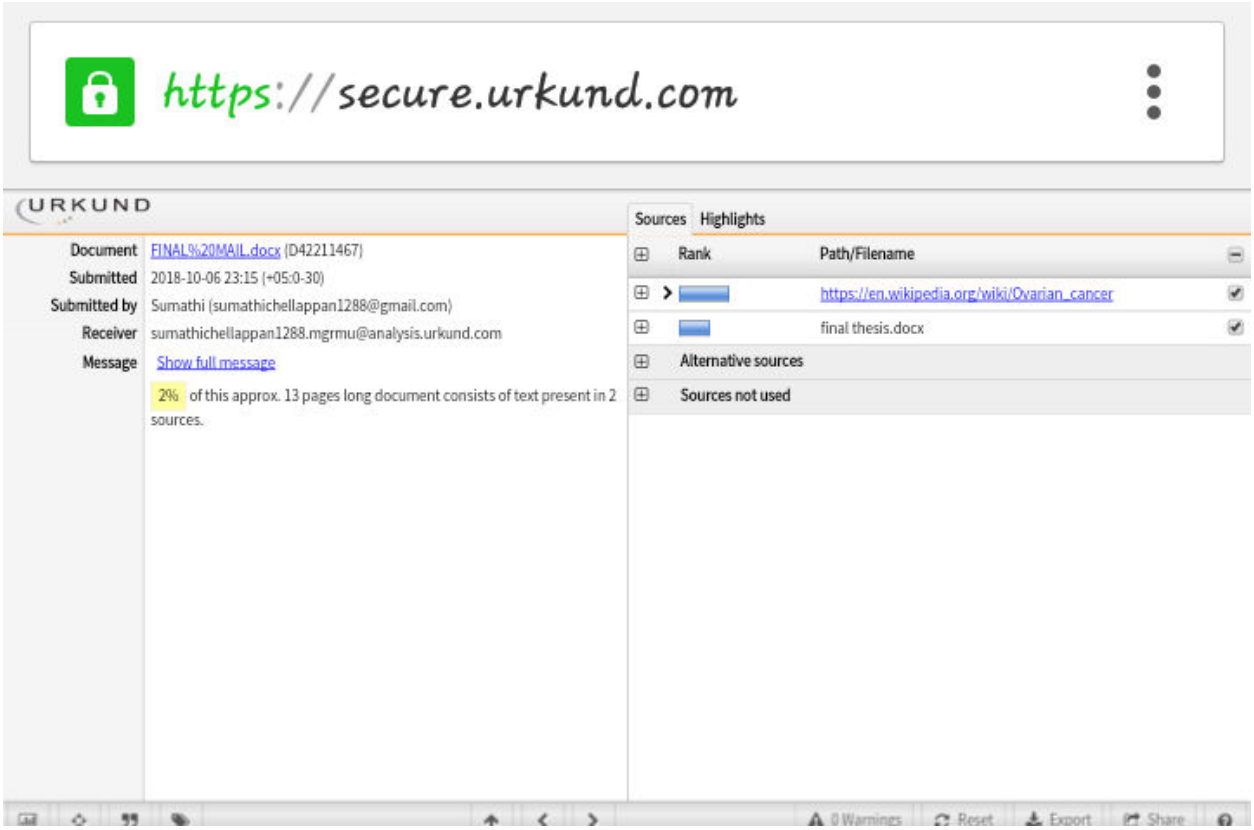
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ABBREVIATIONS

BRCA GENE	- BREAST CANCER GENE
MRI	- MAGNETIC RESONANCE IMAGING
CA 125	- CANCER ANTIGEN
CT	- COMBUTED TOMOGRAPHY
PET	- POSITRON EMISSION TOMOGRAPHY
PAP TEST	- PAPNICOLAOU TEST
HCG	- HUMAN CHORIONIC GONADOTROPHIN
AFP	- ALPHA FETO PROTEIN
TPN	- TOTAL PARENTERAL NUTRITION
HNPCC	- HEREDITARY NON POLYPOSIS COLONIC CANCER
NPV	- NEGATIVE PREDICTIVE VALUE
PPV	- POSITIVE PREDICTIVE VALUE

ABSTRACT

ABSTRACT

Objectives

To compare the effectiveness of risk of malignancy index 1 and 2 to differentiate between benign and malignant ovarian tumors.

Methodology

CA 125 level was done by ECLIA <Enzyme chemi luminescent Immunoassay>. In my study based on ultrasound features, menopausal status, and CA 125 levels RMI 1 & RMI 2 scoring is calculated in women above age 40 years with ovarian tumors. Ultrasound findings were scored with one point for each of the following : mutilocular cyst , evidence of solid areas , evidence of metastasis, presence of ascites, bilateral lesions (zero points for women with no abnormality, one point for women with one abnormality, in women with two or more abnormality 3 points given in RMI 1 , 4 points given in RMI 2). Menopausal status is graded as follows : premenopausal status is graded M = 1 , postmenopausal status is graded as M=3 in RMI 1 , M=4 in RMI 2 .

RESULTS:

In my study RMI 2 has the advantage over RMI 1 to differentiate benign and malignant ovarian tumors. Cut off value to differentiate benign and malignant tumors was 200. Sensitivity and negative predictive value is 100% for both RMI 1 & RMI 2.

Specificity of RMI 2 is 52.5%, for RMI 1 is 47.5%.Positive predictive value of RMI 1 is 32.26%, RMI 2 is 34.48%. Accuracy of RMI 1 IS 58%,for RMI 2 is 62%.

CONCLUSION:

RMI 2 is simple scoring system which is more reliable to identify malignant ovarian tumors. Preoperative evaluation by using this scoring system helps to avoid unnecessary surgical intervention.

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CONTENTS

INTRODUCTION

The ovaries are a pair of integral organs belonging to the female reproductive system. Cancer of the ovary which is common in postmenopausal women, is the fifth most common malignancy affecting women and second most common gynaecological malignancy next to ca endometrium.

However, the most common cause of death from gynaecological malignancy is associated with ovarian carcinoma. If diagnosed and treated early, ovarian malignancies may show a favourable prognosis.

However, the major hurdle faced by gynaecologists is in diagnosing the disease early owing to the fact that ovarian malignancies do not manifest clinically until a later stage, when the prognosis becomes poor. Hence, a high degree of suspicion and multidiagnostic approach is required in patients with nonspecific symptoms in the high risk age group, to detect the malignancy at its earliest.

AIMS & OBJECTIVES

AIM OF THE STUDY:

To identify the ability of RMI 1 and RMI 2 to differentiate between benign and malignant ovarian tumors.

OBJECTIVES OF THE STUDY:

This study is undertaken mainly to identify women at risk of malignant ovarian tumor to avoid unnecessary surgical intervention. Women aged above 40 years admitted with ovarian tumor has included in this study. Three main determinants are menopausal status, ultra sound feature, CA 125 levels.

REVIEW OF LITERATURE

- Morgante et al. in a retrospective study, which revealed that RMI 2 was more reliable with 81% sensitivity and 90% specificity. His study involving 47 patients showed that patients operated on by a gynecological oncologist had a 24% improvement in 5 year survival over those patients operated on by general gynecologists.
- Ulusoy et al. in a study done on 296 women, in which cut off level to differentiate benign and malignant tumor was 153.
- Obeidat et al. in a retrospective study on 100 women with pelvic masses admitted for laparotomy found that RMI diagnosed malignancy more accurately than any single criterion.
- Sharon et al. performed a retrospective review of medical records of 163 patients and found that using a cut-off of 120, the first RMI definition(RMI 1) had 72% sensitivity and 87% specificity; the second (RMI 2) had 76% sensitivity and 81% specificity.

Ovarian cancer is a common, deadly gynecologic malignancy worldwide, usually diagnosed late due to its lack of symptoms and low specificity of screening tests. Among Indian females, ovarian cancer is one of the common cancer to present at an advanced stage. In Japan and Asian countries, 2–6. 5 new cases per 100,000 women per year have been reported. In women over 30 years of age, ovarian carcinoma is the sixth commonest cancer and the fourth commonest cause of death.

Ovarian cancer patients have better outcomes when treated in tertiary centers by a team led by gynecological oncologist. When a prompt referral to such centres is not achieved early, complication rates are higher (~30%) and Survival outcomes are lower (~10%). CA 125, well-known tumor marker used in the risk assessment of ovarian cancer is positive in 80% of patients. CA 125 can be negative in 30% - 50% of patients with stage 1 ovarian cancer

Differentiating benign from malignant ovarian lesions in the absence of surgical exploration and HPE report is not easy. Most of the malignant ovarian tumors diagnosed after menopause. Survival rate in stage 3 is 27% and 16% in stage 4. Most of the ovarian tumors diagnosed at an advanced stage because of its atypical presentation.

USG has 62% sensitivity and 73% specificity to diagnose malignant ovarian tumor.85% epithelial ovarian cancer has elevated CA125 level. RMI scoring system developed to improve the diagnostic quality of ovarian tumors.

In 1990, RMI 1 scoring system was first developed by Jacob et al, it has the sensitivity of 85.4% and specificity of 96.9% to differentiate benign and malignant ovarian tumors.

RMI 2 scoring system was developed by Tingulstad et al in 1996, it was found to be better than RMI 1 to identify benign and malignant ovarian tumors.

If the full surgical staging procedure is carried out initially by a trained gynecological oncologist, women with ovarian malignancy have shown to have better prognosis. A preoperative knowledge of the nature of the adnexal mass is needed to optimally plan surgery at the time of initial treatment. The risk of malignancy index

(RMI) has been shown to be a triage tool with the potential to reduce the workload in a busy gynecological unit.

PATHOLOGY:

Histology of ovarian tumors presents wide variations and may be grouped as follows:

1. Epithelial (80–90%)
2. Non epithelial (10-20%)

RISK FACTORS FOR OVARIAN CANCER

Age - 45-60 years

Nulliparous or low parity

Women with previous PCOS, or on tamoxifen

High calorie, high fat diet

Genetic predisposition – BRCA 1 & 2 genes

Late menopause

Breast and GIT cancer

Prolonged HRT in menopause women

EPITHELIAL CANCERS OF THE OVARY

Histologically

75%-serous

20%- mucinous

2%- endometrioid

1% or less-Brenner tumor, clear cell carcinomas and undifferentiated cancers.

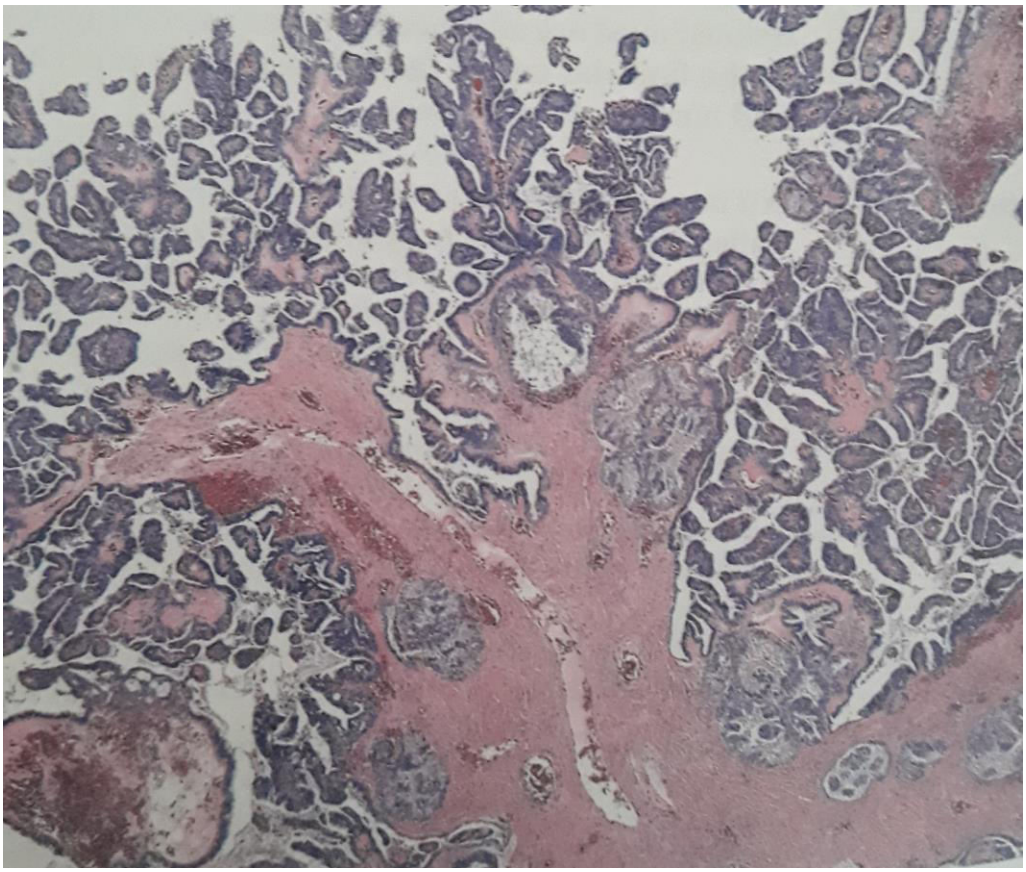
Each tumor type has a histologic pattern similar to a part of the upper genital tract, e.g. serous or papillary pattern -fallopian tube lining, mucinous tumors-endocervical glands and the endometrioid tumors-endometrium.

Nearly 50% benign serous epithelial tumors undergo secondary malignant change, compared to only 5% in mucinous cysts. Borderline tumors has low malignant potential which occur mostly in the premenopausal age group. Invasive cancers common among postmenopausal women which was spread rapidly.

SEROUS CARCINOMA

Stromal invasion is present in malignant serous tumor. Papillary and glandular structures seen in low grade tumor. Mitotic activity and nuclear pleomorphism predominantly seen in malignant serous tumor. Serous psammocarcinoma is a rare variant in which 75% of epithelial nests are associated with psammoma body formation.

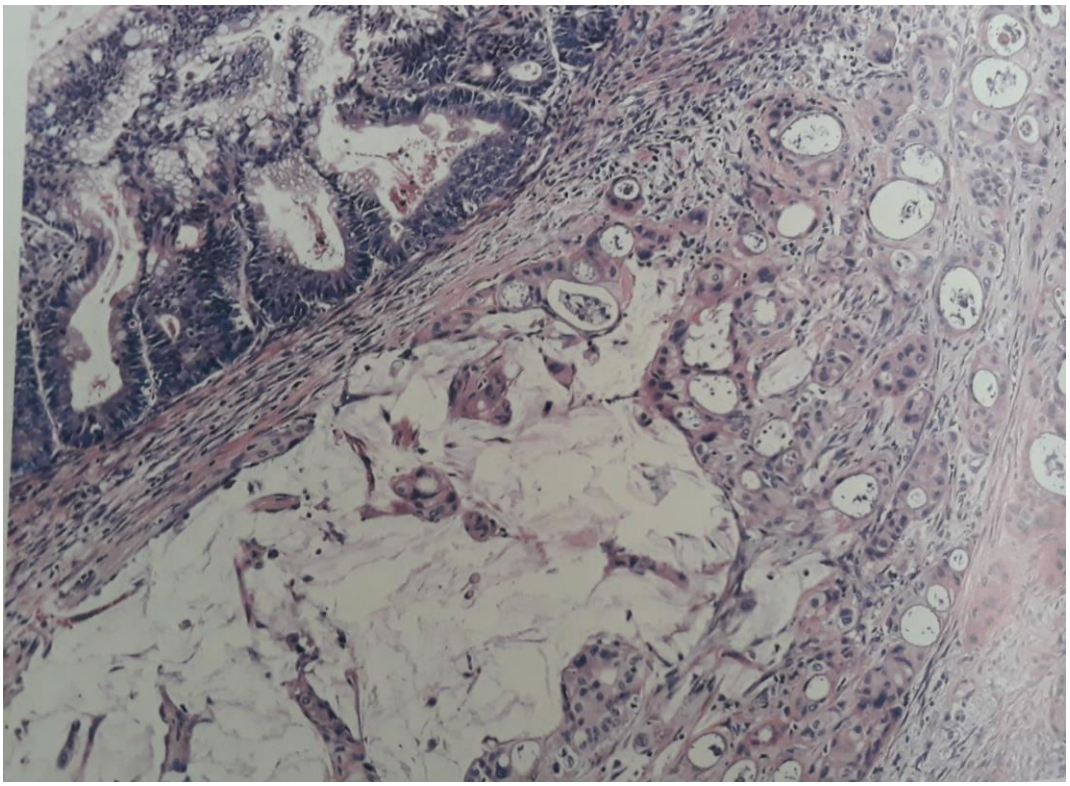
SEROUS TUMOURS OF OVARY



MUCINOUS CARCINOMA

It may grows larger in size , sometimes occupying the entire abdominal cavity. Typical histological features shows loculi which was lined by mucin secreting epithelium.

MUCINOUS TUMOURS OF OVARY



BORDERLINE MUCINOUS TUMORS

This type of tumor was very difficult to diagnose. Multiple sections from many areas in mucinous tumor was taken to diagnose this tumor.

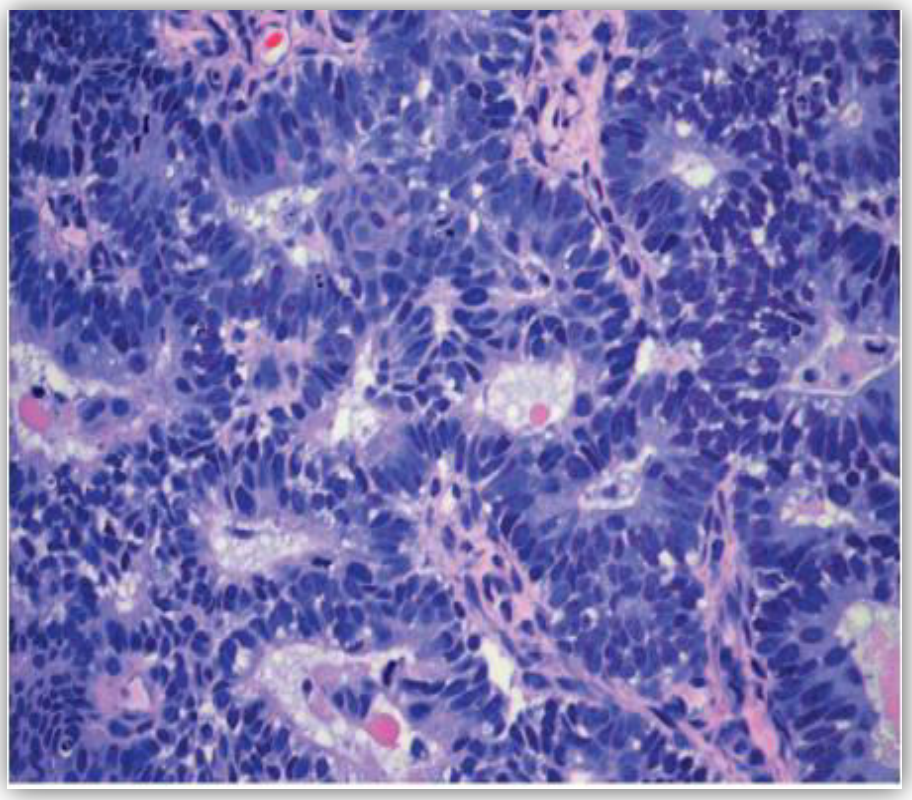
PSEUDOMYXOMA PERITONEI

This tumor is characterized by abundant gelatinous material in the abdominal cavity which was surrounded by fibrous tissue. Most commonly associated with appendiceal mucinous neoplasm and also associated with mucinous ovarian neoplasm.

ENDOMETRIOID TUMORS

It has complex glandular pattern with all the potential variations of epithelium found in the uterus. Borderline endometriod tumor may resemble an endometrial polyp or complex endometrial hyperplasia.

ENDOMETRIOID CARCINOMAS



MULTIFOCAL DISEASE

Around 20% cases of endometriod carcinoma of ovary is associated with endometrial carcinoma. Patients had only 40%-50% survival when metastases from uterus to ovaries is present. Multifocal disease is identified based on the histological pattern of the tumor.

CLEAR CELL CARCINOMAS

Clear cells and hobnail cells are present in the clear cell carcinomas. Focal areas of endometriosis also present.

TRANSITIONAL CELL CARCINOMA

This tumor pattern resembling the transitional cell carcinoma of the urinary bladder. It has good prognosis and sensitive to chemotherapy.

PERITONEAL CARCINOMAS

In this type tumor ovaries are normal or minimally involved. Involvement of uterosacral ligaments, pelvic peritoneum, omentum is predominantly seen. Borderline serous peritoneal tumor has good prognosis. Less common type is peritoneal endometrioid carcinoma. These tumors considered clinically similar as ovarian or fallopian tube cancers.

MESOTHELIOMAS

These tumors appears clinically as multiple intraperitoneal masses. Usually entire peritoneum is involved. It is not related asbestos exposure in women.

CLINICAL FEATURES

80% of epithelial ovarian cancers seen in postmenopausal women. Most of the tumors in postmenopausal women are malignant. Common age group around 56 – 60 years. These cancers rare in less than 45 years of age. Germ cell tumors common among women with age less than 21 years.

PREVENTION

Parity has the inverse relationship with risk of ovarian tumors. Use of OCP reduces the risk of ovarian cancers. OCP use more than 5 years reduce their relative risk to 0.5%. while counselling the patient regarding contraception, have to explain the benefits of OCP. Prophylactic salphingo-oophorectomy reduces the risk of nonuterine pelvic cancers. Ovaries provide protection against cardiovascular disease and osteoporosis.

SCREENING

Screening with transvaginal USG in postmenopausal women is recommended. It has high sensitivity in detecting the early stage ovarian cancers. Trans vaginal colour flow Doppler is not useful for screening. CA 125 is not useful tool for screening but it is used during chemotherapy to monitor the response of the treatment.

GENETIC RISK FOR EPITHELIAL OVARIAN CANCER

Life time risk of ovarian cancer in the US is about 1.4%. Risk is higher in women with strong family history.

HEREDITARY OVARIAN CANCER

BRCA 1 & BRCA 2

Mutation in BRCA 1 gene which is located in chromosome 17, commonly seen in hereditary ovarian cancers. Only few malignancy is associated with BRCA 2 mutation which is located in chromosome 13. BRCA 1 and BRCA 2 associated with both breast and ovarian tumors. Lynch syndrome inherited as autosomal dominant, higher chance of ovarian and endometrial carcinoma. The life time risk of having ovarian tumors in women with BRCA 1 is 28%-44%, which is 27% for in women with BRCA 2 mutation. Ovarian tumors occurs earlier in this syndrome. Breast cancers may be bilateral.

MANAGEMENT OF WOMEN AT HIGH RISK FOR OVARIAN CANCER

Management of ovarian tumors depends on the age, reproductive plans, extent of risk. Histological diagnosis of the family members ovarian cancer should be verified. National institutes of health consensus conference on ovarian cancers recommends, the value of screening with trans vaginal ultrasonography , CA 125 levels, is not established in high risk groups. Most effective way to reduce the risk is doing bilateral salphingo-oophorectomy.

PROPHYLACTIC SALPHINGO-OOPHORECTOMY IN HIGH RISK WOMEN

It reduce the risk of BRCA related gynaecological cancer by 96%. Role of hysterectomy in prevention of ovarian cancers is questionable. Women on tamoxifen benefits from performance of hysterectomy with bilateral salphingo-oophorectomy because tamoxifen intake is associated with risk of benign endometrial lesions.

SYMPTOMS

Most of the women have vague and nonspecific symptoms. Heavy or irregular menstrual bleeding seen in premenopausal age group women. Prseeure symptoms like increased frequency and constipation seen while mass compressing against the the rectum and bladder. Rarely lower abdominal pain and dyspareunia is seen. Other symptoms includes abdominal distension, bloating, constipation, nausea, anorexia, early satiety. Postmenopausal women may presented with vaginal bleeding.

SIGNS

Presence of pelvic mass which solid, fixed and irregular is the most important sign. Any palpable pelvic mass in postmenopausal women is considered to be malignant which is known as the postmenopausal palpable pelvic syndrome.

DIAGNOSIS

To differentiate malignant from benign ovarian masses CA 125 levels are useful. Postmenopausal women with adnexal mass and high serum CA 125 level has PPV of

96% for malignancy. Specificity of the test is low in case of premenopausal women because elevataed CA 125 levels commonly associated with benign conditions.

An interval of atleast two months is allowed, during which hormonal suppression with an OCP may be used. Regression of non neoplastic lesions monitored by pelvic examination and USG. If there is no change in the size of the mass or any increase in size should be considered as neoplastic mass which should be removed by surgery. Most of the bilateral tumors to be malignant. In USG , malignant tumors has the following features like, areas of complexity, irregular borders, multiple echogenic pattern within the mass and dense multiple irregular septae. Better resolution obtained by trans vaginal sonography. Specificity of USG enhanced by colour flow Doppler. Exploratory laparotomy is needed to diagnose the ovarian cancer. Preoperative assessment done to exclude other primary cancers metastatic to the ovary. A Pap test is done ,eventhough it has very low detection rate for ovarian cancer. Endocervical biopsy taken in case of patients with irregular menstrual bleeding or postmenopausal vaginal bleeding.

SPREAD OF THE TUMOR

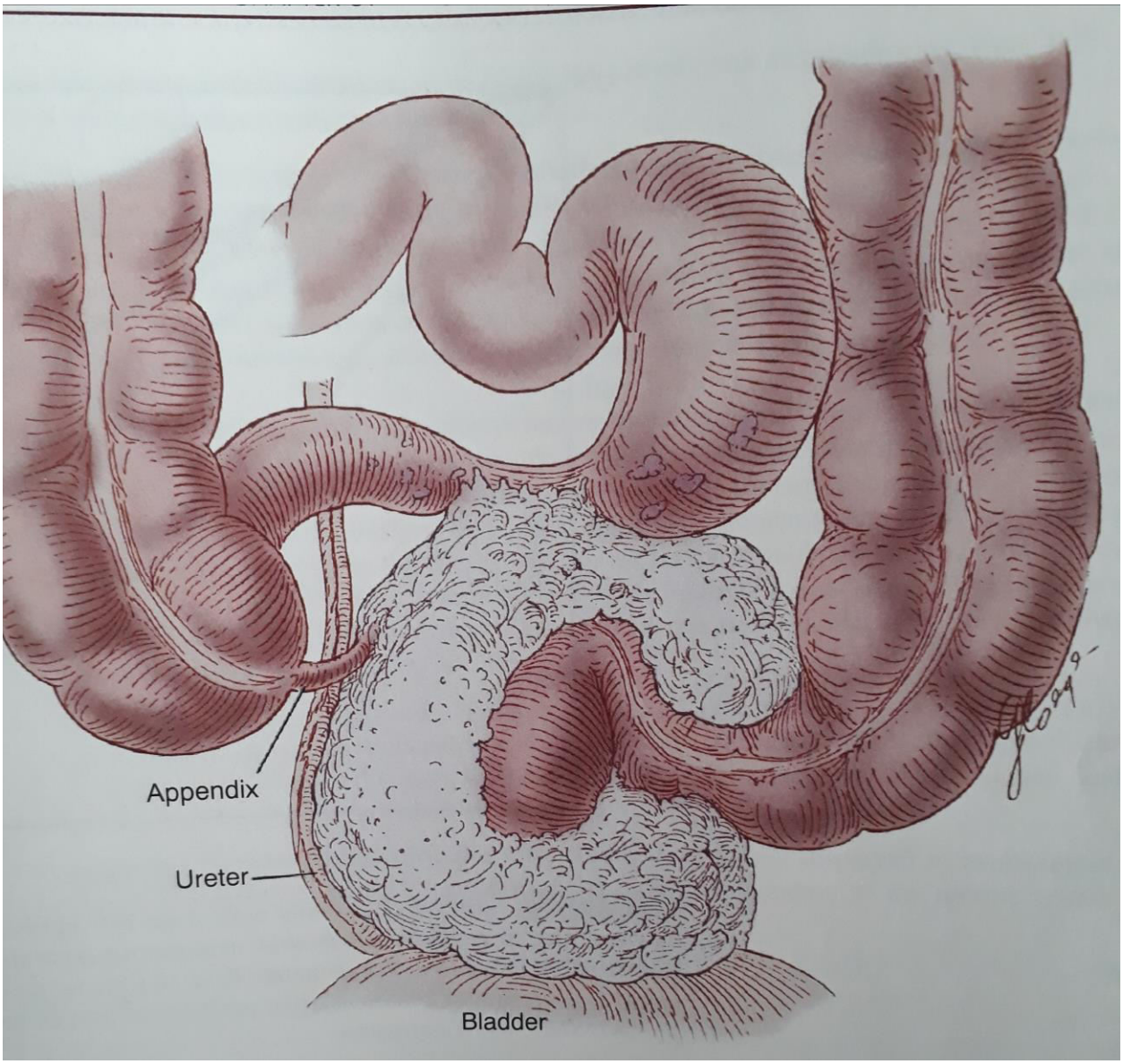
Mode of spread of epithelial ovarian cancers includes haematogenous, lymphatic and exfoliation of cells into the peritoneal cavity.

TRANSCOELOMIC SPREAD

Exfoliation of cells along the surface of the peritoneal cavity is the most common mode of spread of the tumor. Most common sites of metastases includes posterior cul-de-

sac, paracolic gutters, liver capsule, right hemidiaphragm, peritoneal surface of intestine, mesenteries, omentum. Functional intestinal obstruction known as the carcinomatous ileus, in which tumor agglutinates the loops of bowel.

TUMOUR SPREAD TO BLADDER & LARGE INTESTINE



LYMPHATIC SPREAD

In advanced stage disease involvement of pelvic and para aortic lymph nodes is common. Supra clavicular lymph node involvement rarely seen in some conditions.

HEMATOGENOUS SPREAD

Only 2% to 3% of patients had involvement of vital organ parenchyma such as liver and lungs. At the time of diagnosis hematogenous dissemination is uncommon.

NONEPITHELIAL MALIGNANCIES OF THE OVARY

The details of these types are as follows:

1. DYSGERMINOMA
2. TERATOMA

(A) Mature , Dermoid Cyst

(B) Immature— Solid/Cystic

(C) Monodermal teratomas - Struma Ovarii, Carcinoid, Mixed and Others

3. ENDODERMAL SINUS TUMOUR
4. EMBRYONAL CARCINOMA
5. POLYEMBRYOMA
6. CHORIOCARCINOMAS
7. MIXED FORMS.

ENDODERMAL SINUS TUMOR

Endodermal sinus (yolk sac) tumor though rare, is the second most common of germ cell origin originating from a multipotentia embryonal tissue,with selective differentiation of yolk sac elements. This is why the tumor is rich in alpha-fetoproteins and alpha-l-antitrypsin. Histologically, it presents with papillary projections composed of a central core of blood vessels enveloped by immature epithelium. Intracellular and extracellular hyaline droplets are present in all tumors. The alpha-fetoprotein content can be stained by immune peroxidase techniques. Most of these patients are children or young women, presenting with abdominal pain and a pelvic mass. They are rapidly growing tumors. Although they are highly malignant, they respond to chemotherapy with good survival rate.

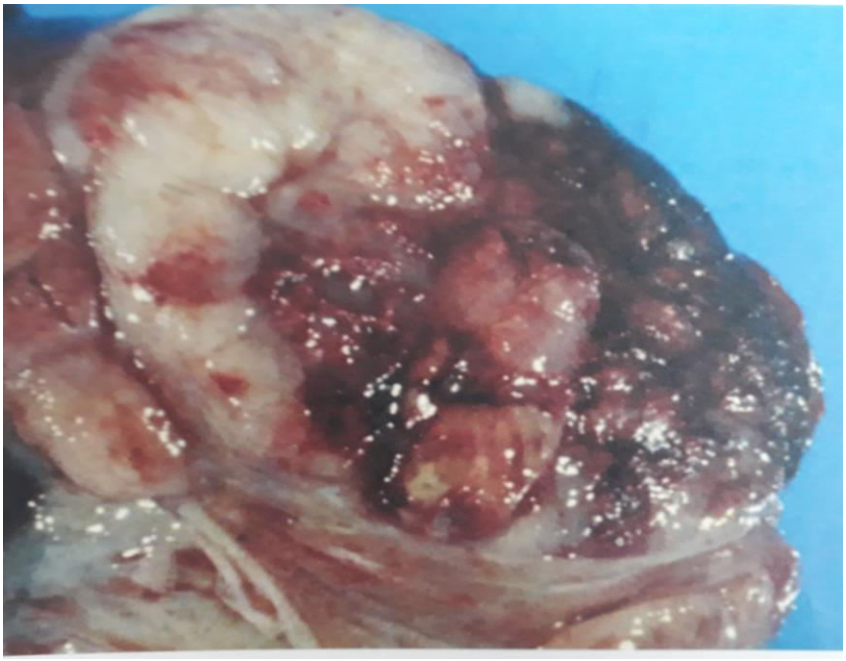
CHORIOCARCINOMA

Part of a mixed germ cell tumor. Its origin as a teratoma can be confirmed in prepubertal girls, when the possibility of its gestational origin can be definitely excluded. The tumors have high vascularity. Histologically, it shows a dimorphic population of syncytiotrophoblasts and cytotrophoblasts. It secretes large quantities of human chorionic gonadotropin, which forms an ideal tumor marker in the diagnosis and management of the tumor. The tumor is highly malignant, and metastasizes by blood stream to the lungs, brain, bones and other viscera.

EMBRYONAL CELL CARCINOMA

Embryonal cell carcinoma is a rare tumor accounting for about 5% of all germ cell tumors, and occurring in prepubertal girls, secretes both alpha-fetoproteins and chorionic gonadotropins. It is associated with symptoms of precocious puberty and menstrual irregularities. It is highly malignant. The condition may be associated with fever due to torsion, rupture and haemorrhage. Although 20–25% of all ovarian neoplasms are germ cell tumors, only 3–5% are malignant. Dysgerminoma and pure germinomas secrete lactose dehydrogenase. Dysgerminomas are highly radiosensitive. They also respond well to chemotherapy without interfering with future fertility and therefore chemotherapy is preferred.

DYSGERMINOMA OF OVARY



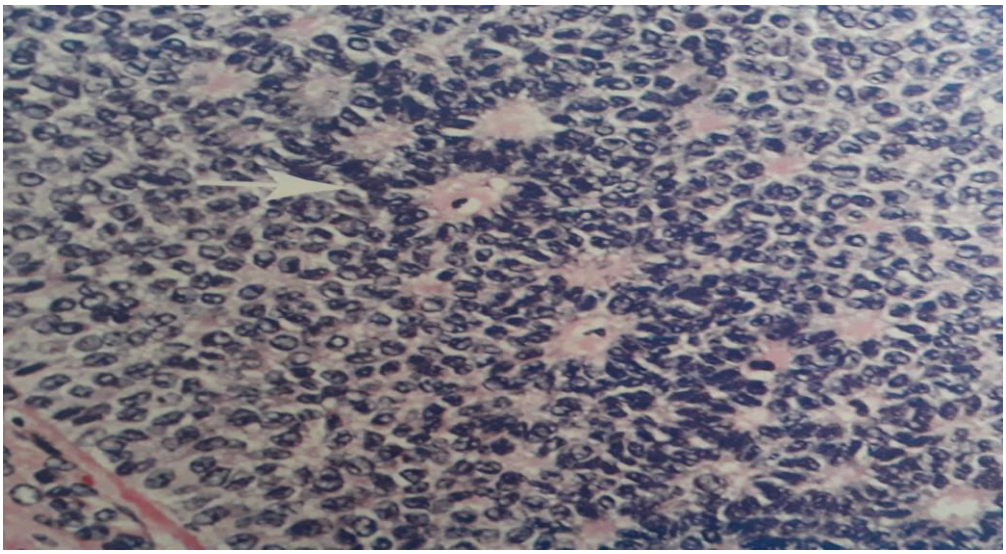
SEX CORD STROMAL TUMORS

Sex cord stromal tumors are either benign or malignant. They account for about 5–8% of all ovarian malignancies. These tumors are composed of various combinations of cells consisting of granulosa cells, theca cells, sertoli and leydig cells as well as morphologically indifferent cells. They are also called mesenchymomas.

GRANULOSA CELL TUMORS

Granulosa cell tumors secrete estrogens. Depending on the age of their appearance, they may cause precocious puberty, menometrorrhagia and episodes of abnormal uterine bleeding are common in women of childbearing age and postmenopausal bleeding in elderly women. Endometrial hyperplasia occurs in 25–50% of patients, and endometrial carcinoma occurs in about 5% of cases, more likely with theca cell tumor. A granulosa cell tumor secretes inhibin, which is a marker for this tumor.

GRANULOSA CELL TUMOUR



SERTOLI LEYDIG CELL TUMORS

Androblastomas or arrhenoblastomas commonly occurring in the third and fourth decades of life are very rare and account for 0.2% of all ovarian neoplasms. They secrete androgens which can cause defeminization followed by masculinization. The women experiences oligomenorrhoea followed by amenorrhoea, flattening of the breasts, acne, hirsutism, enlargement of the clitoris and finally a change in voice. On removal of the tumor, all the above changes reverse except voice change.

SARCOMA

Ovarian sarcomas are rare. Many tumors labelled as sarcomas have been misdiagnosed histologically and in reality, they are granulosa cell tumors or anaplastic

carcinomas. Sarcomas arise most frequently after menopause, particularly in multipara. They give rise to multiple metastases.

MANAGEMENT

The operable cases (Stages I and II) should undergo total hysterectomy and bilateral salpingo-oophorectomy with omentectomy.

Advanced,inoperable case (Stages III and IV) will benefit from debulking surgery and tumor removal. Postoperative chemoradiation improve the survival and quality of life. Surgery provides symptomatic relief and reduces the amount of malignant tissue to be subjected to chemotherapy .Smaller residual tissue responds better to chemotherapy and thus remission period and survival is improved. Pre-operative cisplatin followed by surgery is lately employed.

Postoperative chemoradiation depend upon the staging and type of tumor . The duration of chemotherapy is decided by the level of tissue markers.

INTERVAL SURGERY

Some advanced and bulky tumors are initially treated with chemotherapy for 3 cycles,followed by a debulking surgery and a postoperative chemotherapy as dictated by tissue marker.

Laparoscopic surgery, if undertaken, may have the following disadvantages:

1. Possibility of spillage during surgery with recurrence.

2. Port-site metastasis in 1–1.5% cases,which can be reduced by use of endospecimen bag, lavage and use of intraperitoneal chemotherapy.

SECOND LOOK SURGERY

1. To detect the presence of any residual tumor following a planned course of chemotherapy and decide if further chemotherapy is required. The availability of tissue markers for vast majority of ovarian tumors in the follow-up, have decreased the importance of second-look surgery and surgical morbidity is also eliminated. Besides, microscopic residual tumors may not be detected at laparoscopy.

2. Following a 3–6 month course of chemotherapy in an inoperable case, second-look surgery may enable TAH and BSO or debulking procedure.

3. In a recurrent tumor.

4. Instead of laparotomy, second-look laparoscopy is another alternative.

Combination of surgery, radiotherapy and chemotherapy has enhanced the survival rate and quality of life considerably.

Recurrent tumor –

Treatment modality in case of recurrent tumour is based upon the type of tumor, size and its histology.

- Second-look surgery and removal of the lesion—for a single-site recurrence.
- Chemotherapy—for visceral metastasis.

- Radiotherapy—preferably for nodal metastasis.

Intraperitoneal chemotherapeutic drug may be instilled in a small residual tumor, at the end of surgery.

Stem cell therapy may have a role in future. Dysgerminoma and granulosa cell tumor respond well to both chemotherapy and radiotherapy. In a young woman, fertility-retaining surgery of unilateral ovariotomy is followed by chemotherapy as radiotherapy destroys the other ovary. In older woman,radiotherapy can be given following hysterectomy and salpingo-oophorectomy.

PRIMARY CHEMOTHERAPY

Advanced malignancy is considered relatively sensitive to cytotoxic agents and the duration of survival among patients has increased over the past two decades, yet, fewer than 20 percent can be cured.

INTRAVENOUS CHEMOTHERAPY

Platinum-based chemotherapy is the mainstay systemic treatment of ovarian cancer. The most widely used regimen in the US is six courses of carboplatin and paclitaxel. Additional cycles required to achieve clinical remission suggests relative

tumor chemoresistance and usually leads to an earlier relapse. Addition of a third cytotoxic agent has been reported to further improve outcome.

INTRAPERITONEAL CHEMOTHERAPY

In January 2006, the national cancer institute issued a rare clinical announcement encouraging the use of intraperitoneal chemotherapy. The median duration of overall survival was 66 months in the iv/ip group compared with 50 months in the intravenous treatment group. Despite this dramatic improvement in survival, many clinicians still consider ip chemotherapy to be an experimental treatment.

MANAGEMENT OF PATIENTS IN REMISSION

In most women with advanced ovarian cancer, surgery plus platinum-based chemotherapy will result in clinical remission. However, up to 80 % will relapse eventually and die from disease progression. Lower CA125 levels generally are associated with fewer relapses and longer survival. Since most patients achieving remission will have residual, clinically occult, drug-resistant cells, several options are appropriate to consider, but there is no concrete proof that any intervention is beneficial.

SURVEILLANCE

After completion of treatment, patients should be regularly followed up with examinations and CA125 determinations. To monitor advanced ovarian cancer patients,

imaging tests may be indicated more frequently and a heightened suspicion for relapse should be maintained.

RADIATION THERAPY

In the US, when compared to Europe, patients in remission after primary therapy are rarely treated with whole abdominal radiotherapy due to doubtful benefit and fear of excessive toxicity. However, the long-term effectiveness of this consolidation strategy is comparable with that achieved with other modalities of treatment. As a result, it may be considered for selected patients with microscopic disease detected at second-look surgery.

MANAGEMENT OF RECURRENT OVARIAN TUMOR

Gradual elevation in levels of CA125 is usually the first sign of relapse. In such cases tamoxifen may be administered frequently since it has some activity in treating recurrent disease with minimal toxicity. Without treatment, the recurrence usually will become obvious clinically within 2 to 6 months, mostly within the abdomen. Women who progress during primary chemotherapy are classified as having platinum-refractory disease .Those who relapse within 6 months have platinum-resistant ovarian cancer. Patients in either category have a poor prognosis, and palliative nonplatinum chemotherapy is effectively the only option. Women who relapse more than 6 to 12 months after completion of primary therapy are considered platinum-sensitive.

PALLIATION OF END STAGE OVARIAN CANCER

During treatment, intermittent episodes of partial small and large bowel obstruction are common, which may be worse in those with recurrent disease.Bowel obstruction that does not resolve with nasogastric suction can be managed aggressively with surgical intervention, initiation of total parenteral nutrition (TPN), and continued chemotherapy. Ideally, a colostomy, ileostomy, or intestinal bypass will return reasonably normal bowel function. Unfortunately, a satisfactory surgical result is often not possible because of multiple sites of partial or complete obstruction. In addition, successful palliation is rarely achieved when the transit time is prolonged by diffuse peritoneal carcinomatosis or when the anatomy requires a bypass that results in the short bowel syndrome. Further, recovery may be complicated by an enterocutaneous fistula, reobstruction, or other morbid event. For some patients, a refractory bowel obstruction can be managed by placement of a palliative gastrostomy tube, IV hydration, and hospice care. In a woman with symptomatic, rapidly reaccumulating ascitic fluid, repeated paracenteses or placement of an indwelling peritoneal catheter will provide symptomatic relief. Similarly, a refractory malignant pleural effusion can be managed by thoracentesis, pleurodesis, or placement of indwelling pleural catheter. Although these procedures and others may be appropriate in selected patients, the inability to halt disease progression should be acknowledged. In addition, any intervention has the potential to result in an unanticipated catastrophic complication. Overall, palliative procedures are used most compassionately when incorporated into the overall treatment plan.

MATERIALS AND METHODS

SOURCE OF DATA:

A total of 50 women who attended the gynecology clinic of the department of obstetrics and gynecology at Coimbatore medical college hospital as well as those admitted in the ward were selected for the study.

STUDY PERIOD:

June 2017 TO June 2018.

STUDY DESIGN:

Comparative Prospective study.

STUDY SUBJECTS:

Sample size: 50

INCLUSION CRITERIA:

1. Women aged above 40 years

2. Women presenting as adnexal mass and admitted for evaluation and treatment

EXCLUSION CRITERIA:

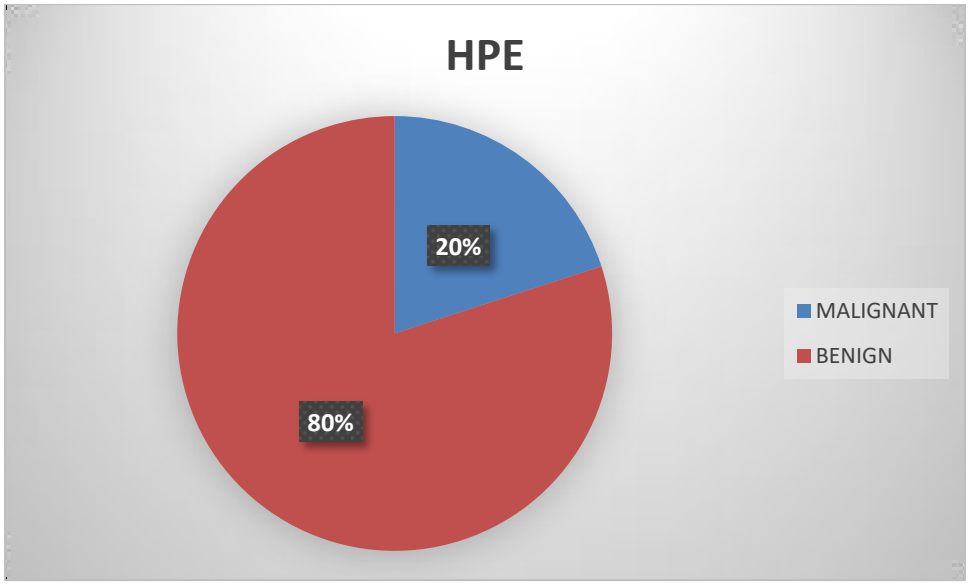
1. Age less than 40 years

2. Women already diagnosed of ovarian malignancy and received chemotherapy for the same

OBSERVATIONS

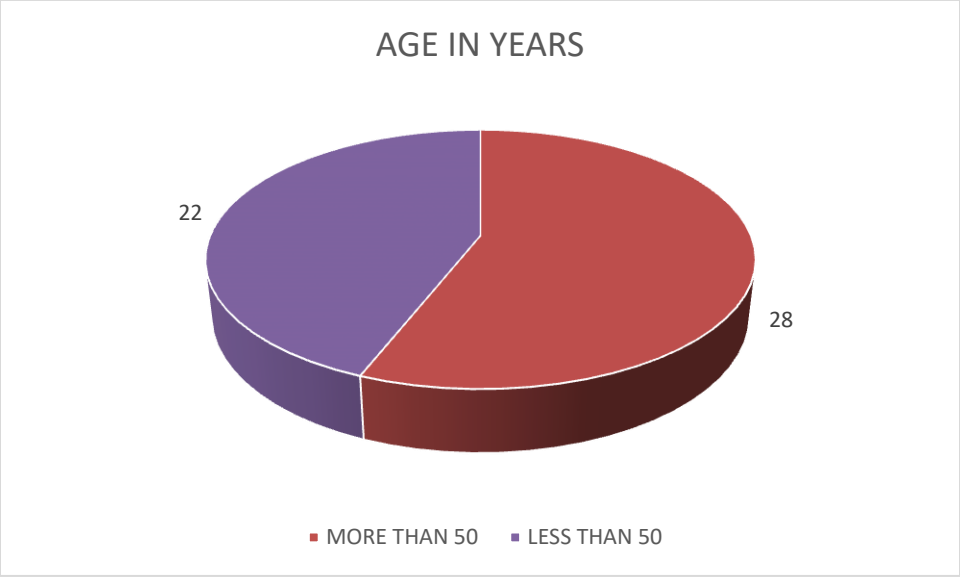
RESULTS

HISTOPATHOLOGY EXAMINATION		
	NO OF PATIENTS	PERCENTAGE
MALIGNANT	10	20%
BENIGN	40	80%

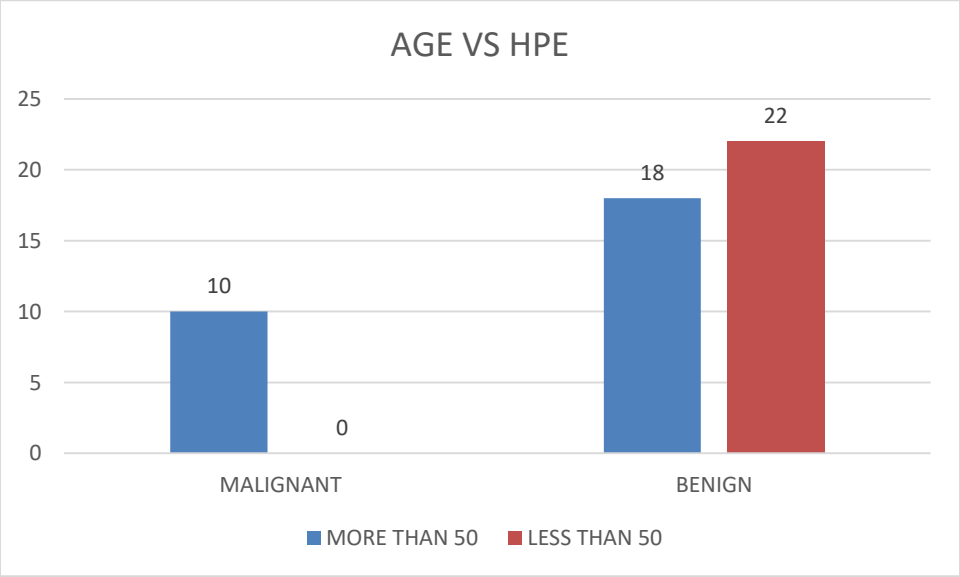


AGE DISTRIBUTION

AGE IN YEARS	NO OF PATIENTS	PERCENTAGE
MORE THAN 50	28	56%
LESS THAN 50	22	44%



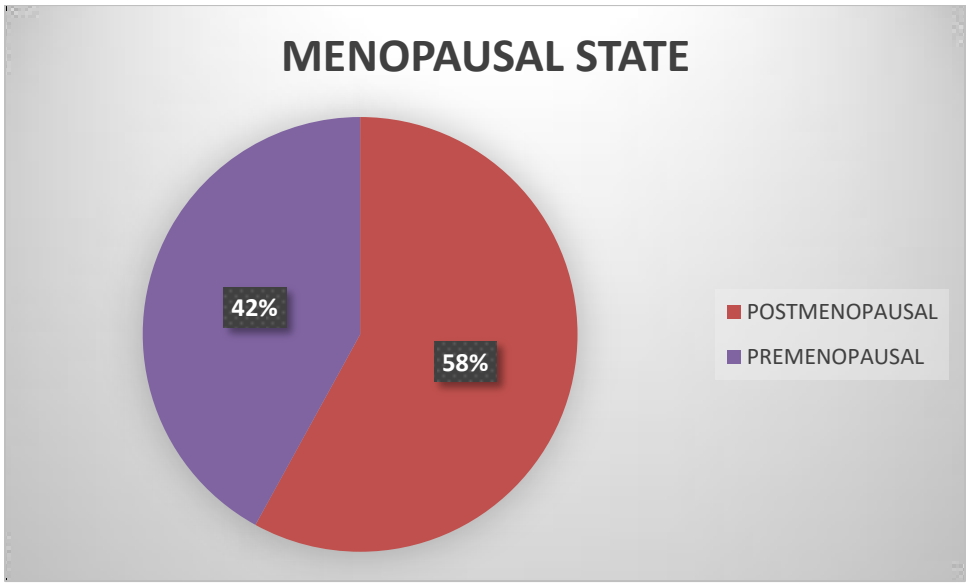
AGE VS HPE		
AGE IN YEARS	MALIGNANT	BENIGN
MORE THAN 50	10	18
LESS THAN 50	0	22
CHI SQUARE TEST		
P VALUE - 0.002		
SIGNIFICANT		



In my study out of 50 patients women with more than 50 years, 10 patients had malignant ovarian tumors. P value is 0.002.

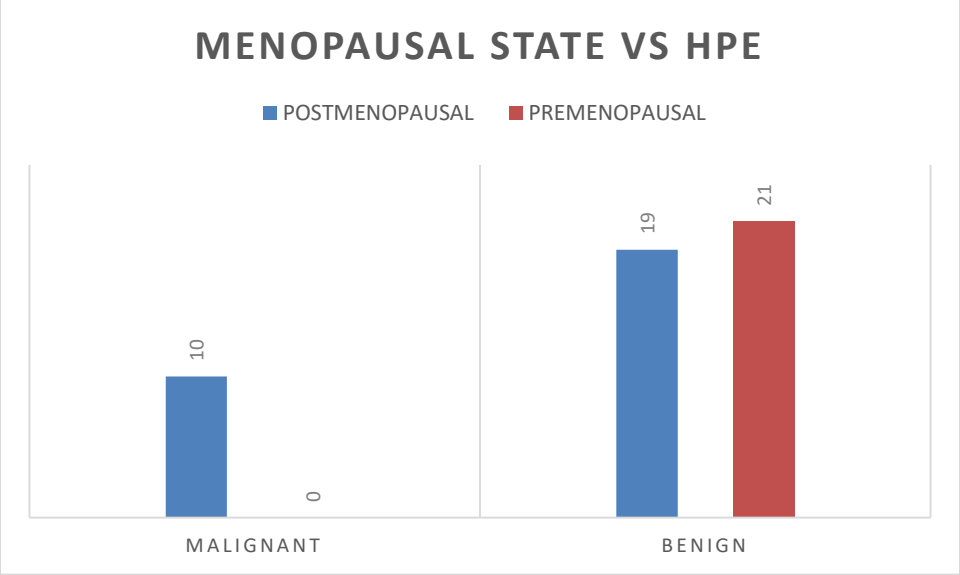
MENOPAUSAL STATE

MENOPAUSE	NO OF PATIENTS	PERCENTAGE
POSTMENOPAUSAL	29	58%
PREMENOPAUSAL	21	42%



MENOPAUSAL STATE VS HPE		
MENOPAUSE	MALIGNANT	BENIGN
POSTMENOPAUSAL	10	19
PREMENOPAUSAL	0	21
CHI SQUARE TEST		
P VALUE - 0.003		
SIGNIFICANT		

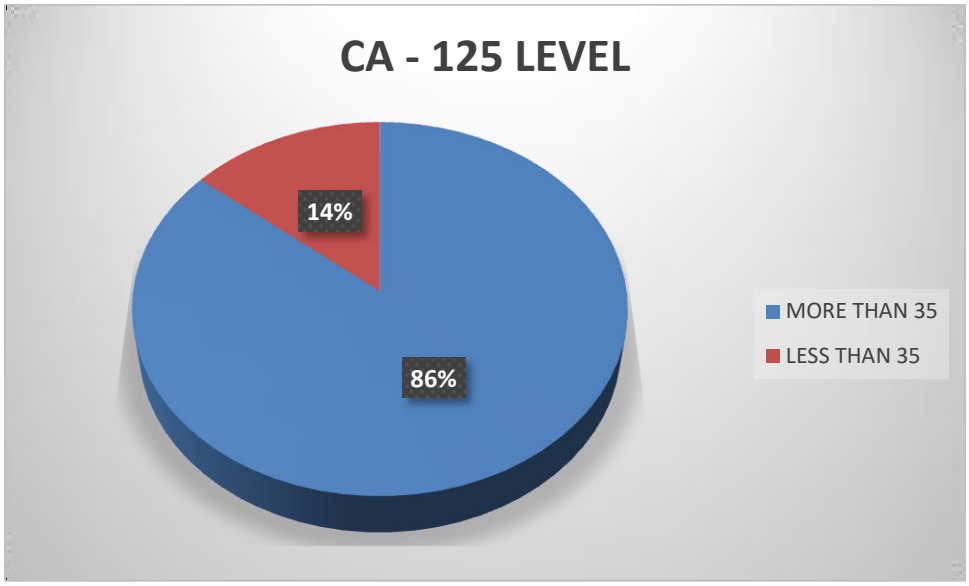
This picture depicts relationship between menopausal status and HPE correlation of ovarian tumors.



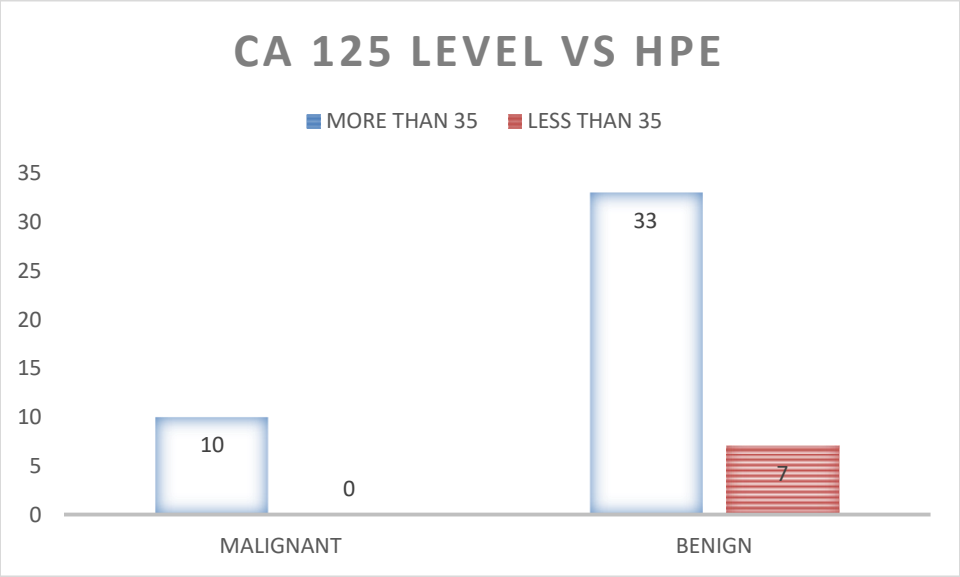
Out of 50 patients 10 postmenopausal women had malignant ovarian tumor. Significant p value is 0.003.

CA – 125 LEVELS

CA – 125	NO OF PATIENTS	PERCENTAGE
MORE THAN 35	43	86%
LESS THAN 35	7	14%

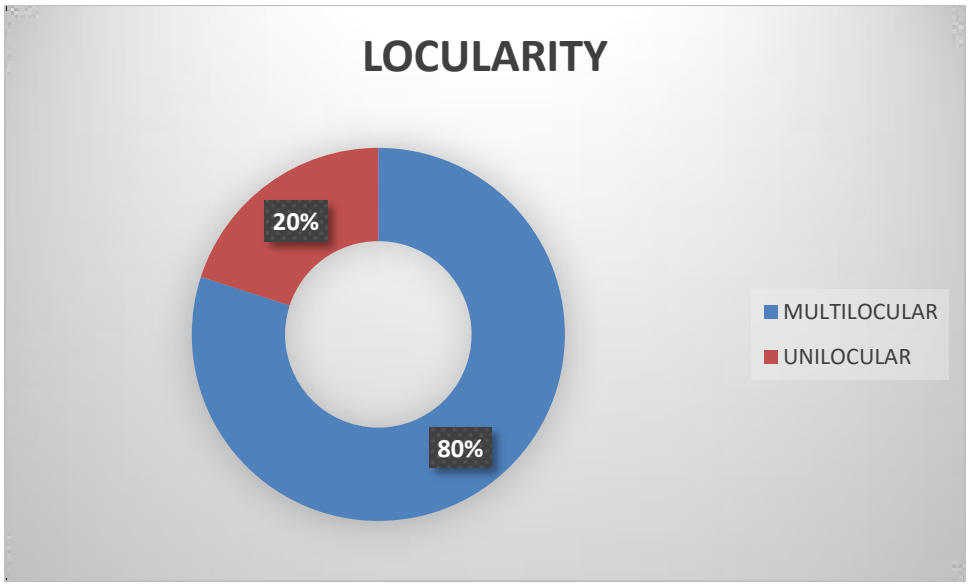


CA -125 LEVEL VS HPE		
CA - 125	MALIGNANT	BENIGN
MORE THAN 35	10	33
LESS THAN 35	0	7
CHI SQUARE TEST		
P VALUE - 0.154		
NON SIGNIFICANT		

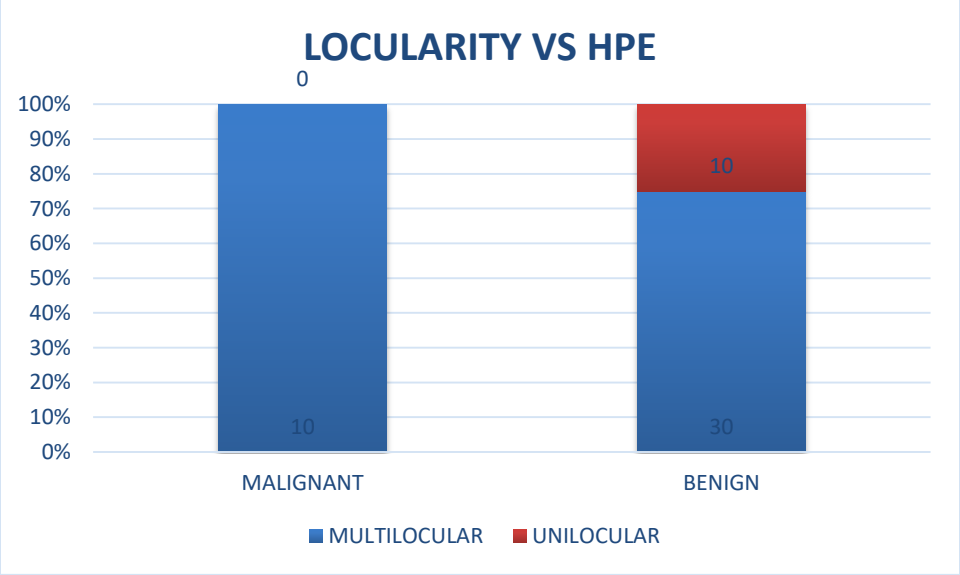


LOCULARITY

LOCULARITY	NO OF PATIENTS	PERCENTAGE
MULTILOCULAR	40	80%
UNILOCULAR	10	20%

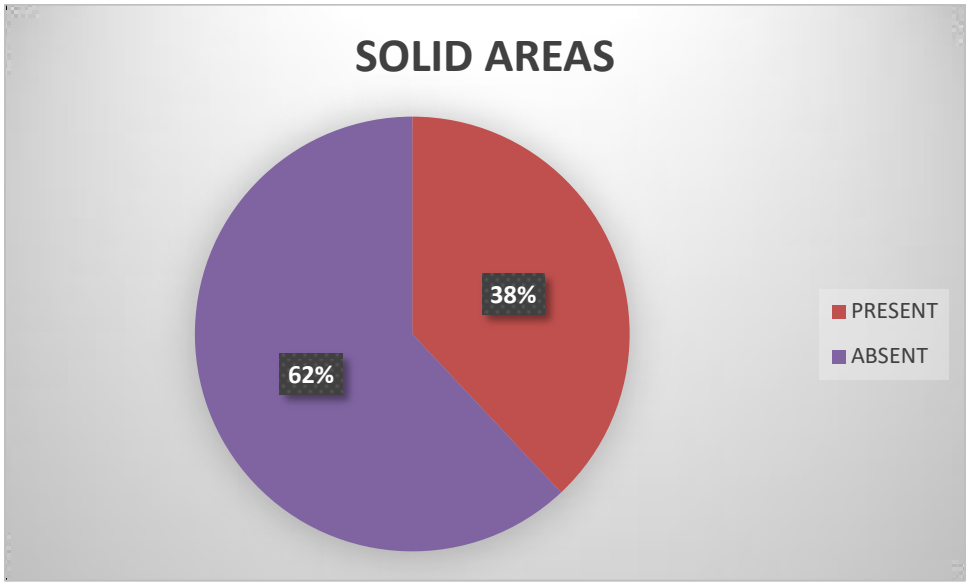


LOCULARITY VS HPE		
LOCULARITY	MALIGNANT	BENIGN
MULTILOCULAR	10	30
UNILOCULAR	0	10
CHI SQUARE TEST		
P VALUE - 0.077		
NON SIGNIFICANT		

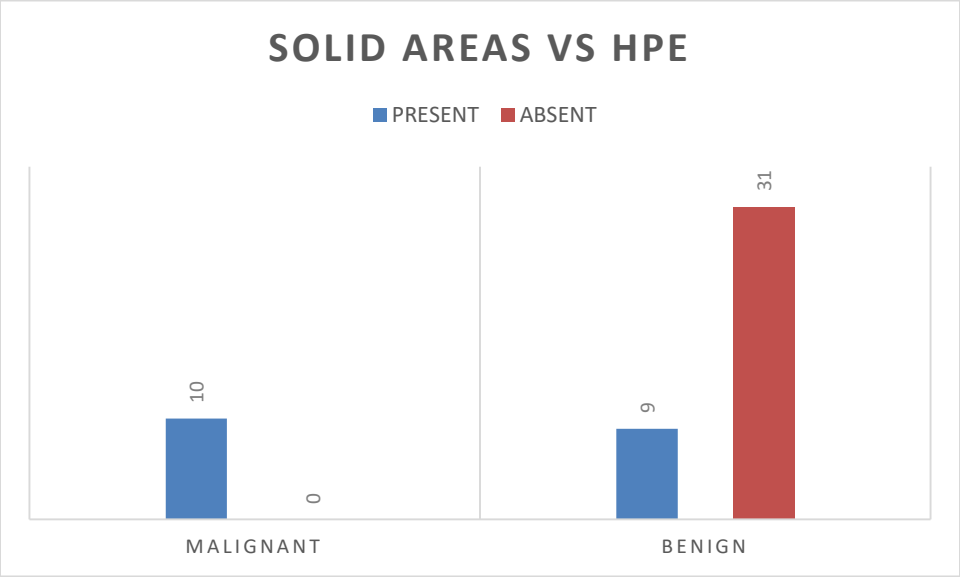


SOLID AREAS

SOLID AREAS	NO OF PATIENTS	PERCENTAGE
PRESENT	19	38%
ABSENT	31	62%



SOLID AREAS VS HPE		
SOLID AREAS	MALIGNANT	BENIGN
PRESENT	10	9
ABSENT	0	31
CHI SQUARE TEST		
P VALUE - 0.001		
SIGNIFICANT		

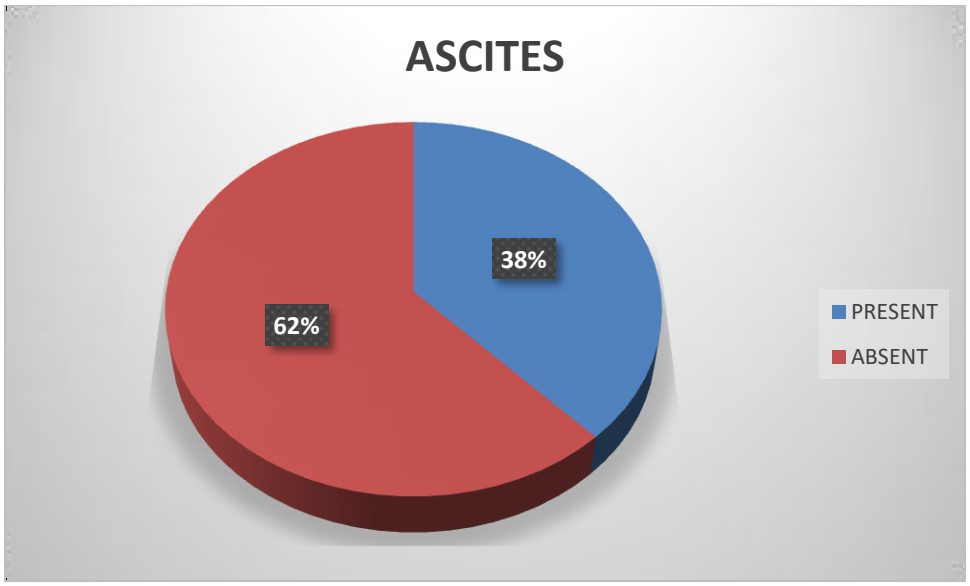


In ovarian tumors with solid areas in ultrasonography 10 patients had malignancy when it is compared with histopathological examination reports. Significant

P value is 0.001.

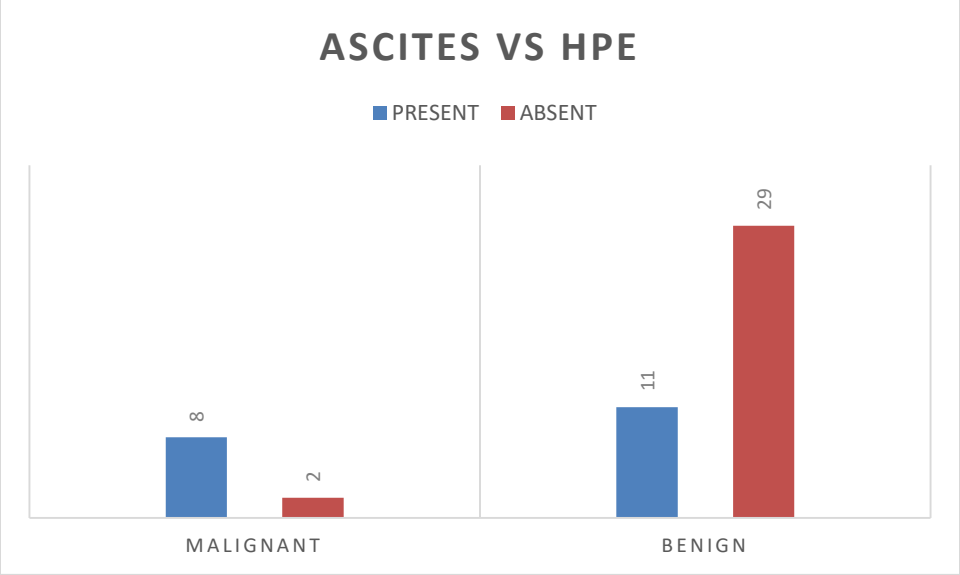
ASCITES

ASCITES	NO OF PATIENTS	PERCENTAGE
PRESENT	19	38%
ABSENT	31	62%



This table compares relationship of malignant tumors in patients with ascites in comparison to histopathological reports.

ASCITES VS HPE		
ASCITES	MALIGNANT	BENIGN
PRESENT	8	11
ABSENT	2	29
CHI SQUARE TEST		
P VALUE - 0.002		
SIGNIFICANT		

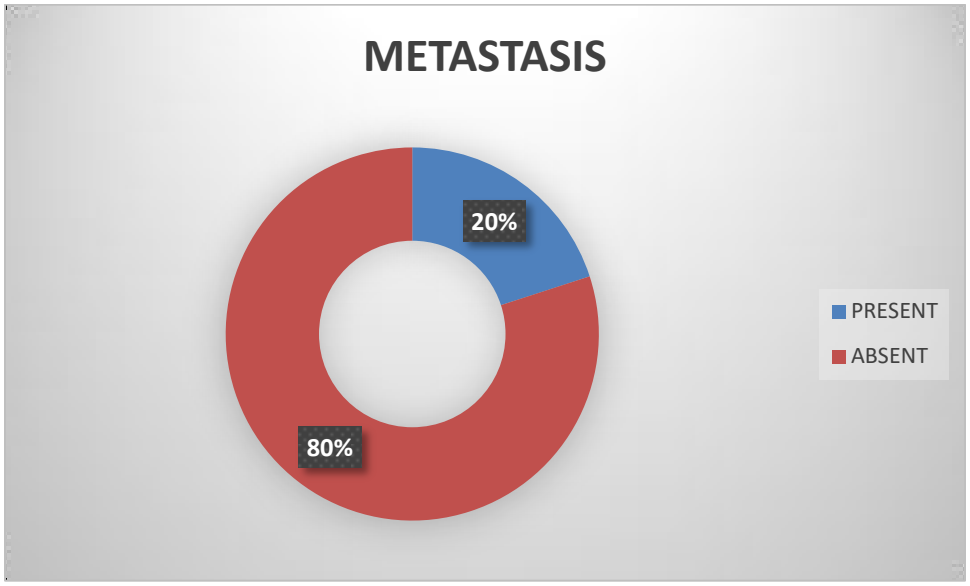


In my study 8 patients with ascites histopathological report positive for malignancy.

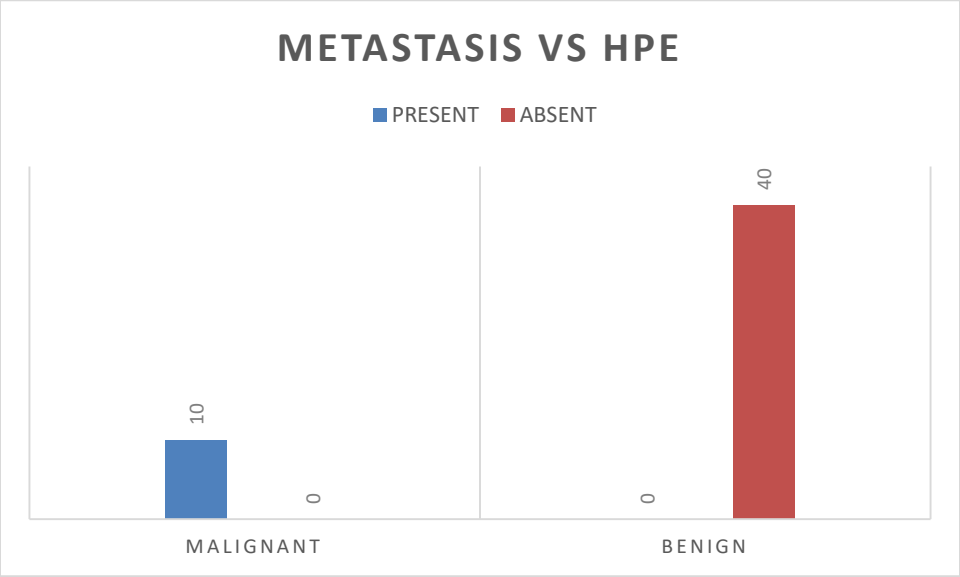
Significant p value is 0.002.

METASTASIS

METASTASIS	NO OF PATIENTS	PERCENTAGE
PRESENT	10	20%
ABSENT	40	80%



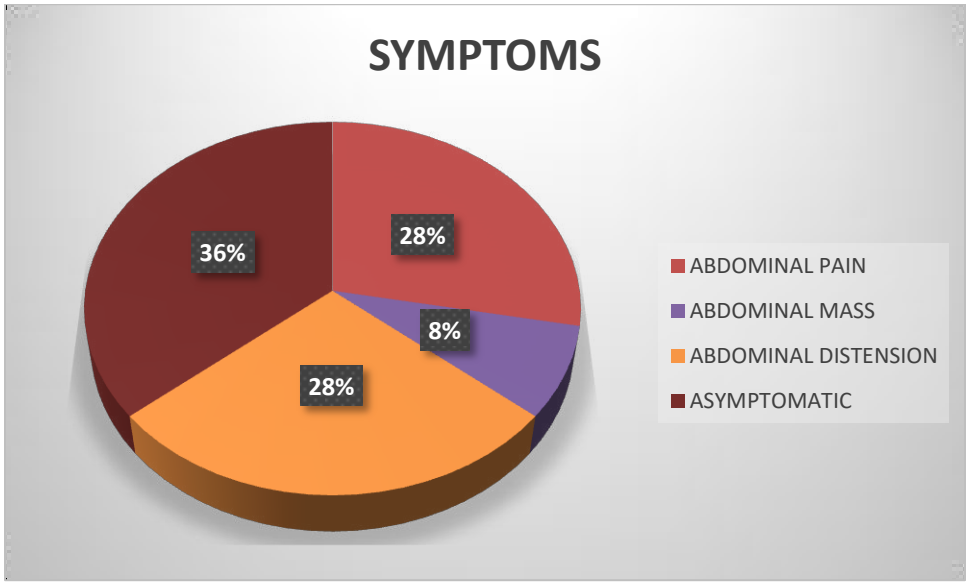
METASTASIS VS HPE		
METASTASIS	MALIGNANT	BENIGN
PRESENT	10	0
ABSENT	0	40
CHI SQUARE TEST		
P VALUE - 0.000		
SIGNIFICANT		



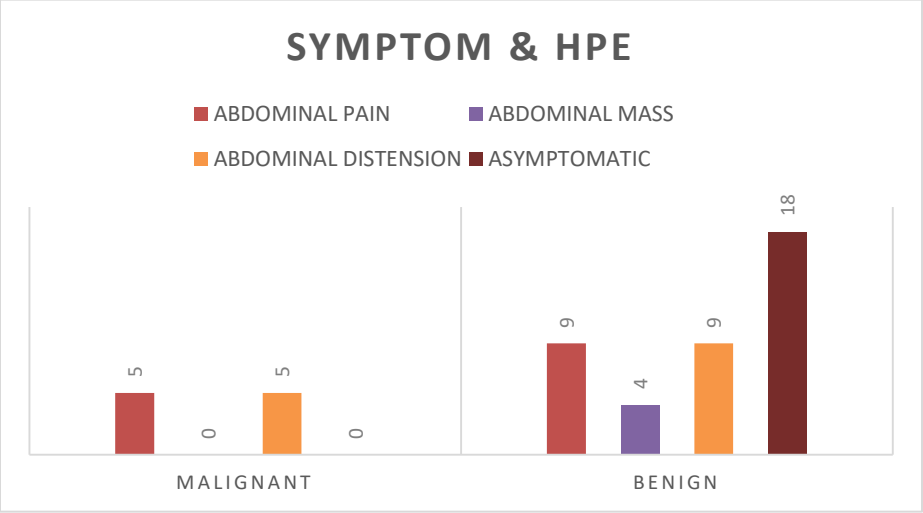
Out of 50 patients, 10 patients with metastasis, histopathology reports positive for malignancy. P value is 0.000.

SYMPTOMS

SYMPTOMS	NO OF PATIENTS	PERCENTAGE
ABDOMINAL PAIN	14	28%
ABDOMINAL MASS	4	8%
ABDOMINAL DISTENSION	14	28%
ASYMPTOMATIC	18	36%



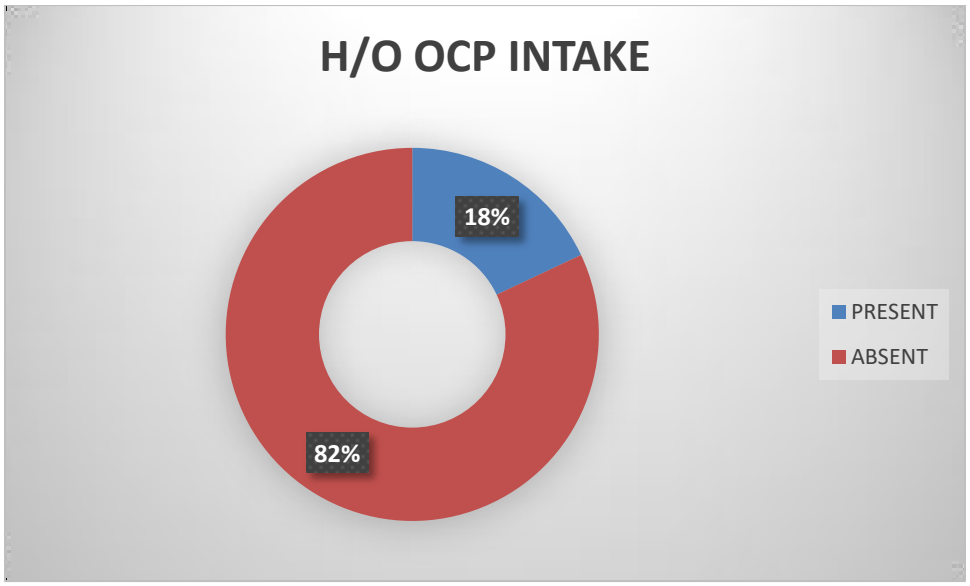
SYMPTOMS VS HPE		
SYMPTOMS	MALIGNANT	BENIGN
ABDOMINAL PAIN	5	9
ABDOMINAL MASS	0	4
ABDOMINAL DISTENSION	5	9
ASYMPTOMATIC	0	18
KRUSKAL WALLIS TEST		
P VALUE - 0.020		
SIGNIFICANT		



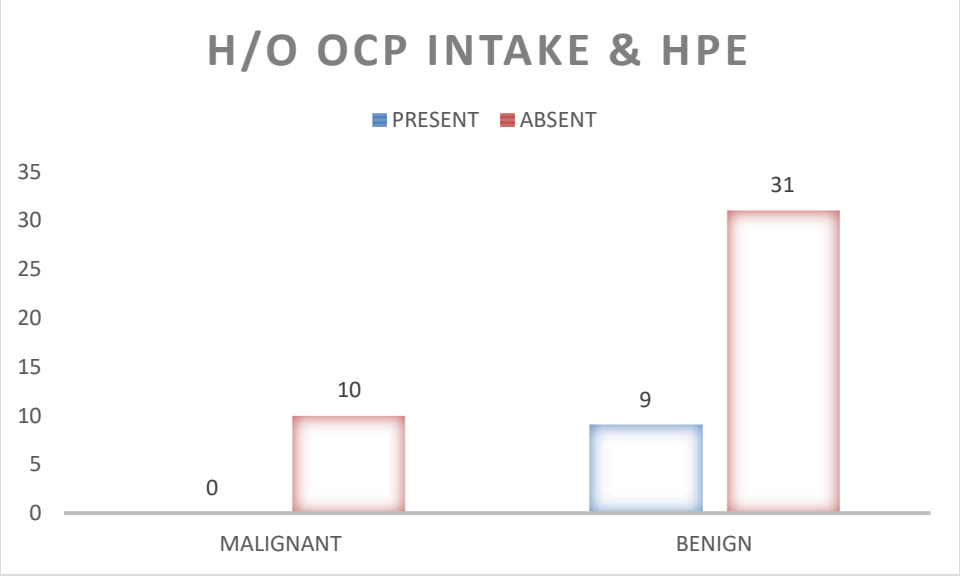
Out of 50 patients, 5 patients with abdominal pain and 5 patients with abdominal distension, positive for malignant ovarian tumor. Significant p value is 0.020.

HISTORY OF OCP INTAKE

H/O OCP INTAKE	NO OF PATIENTS	PERCENTAGE
PRESENT	9	18%
ABSENT	41	82%



H/O OCP INTAKE VS HPE		
H/O OCP INTAKE	MALIGNANT	BENIGN
PRESENT	0	9
ABSENT	10	31
CHI SQUARE TEST		
P VALUE - 0.098		
NON SIGNIFICANT		

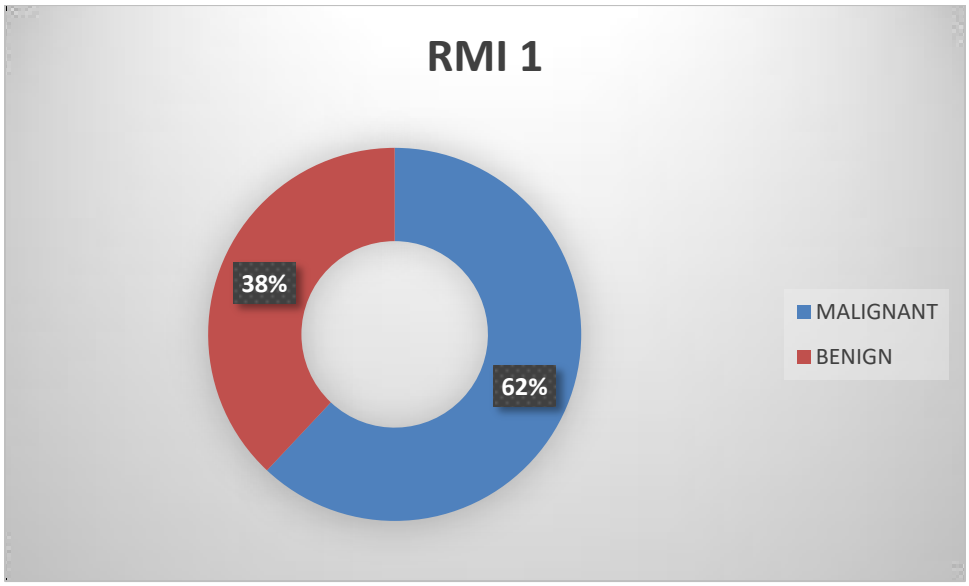


In my study there is no significant relationship between oral contraceptive pills intake and incidence of malignant ovarian tumors.

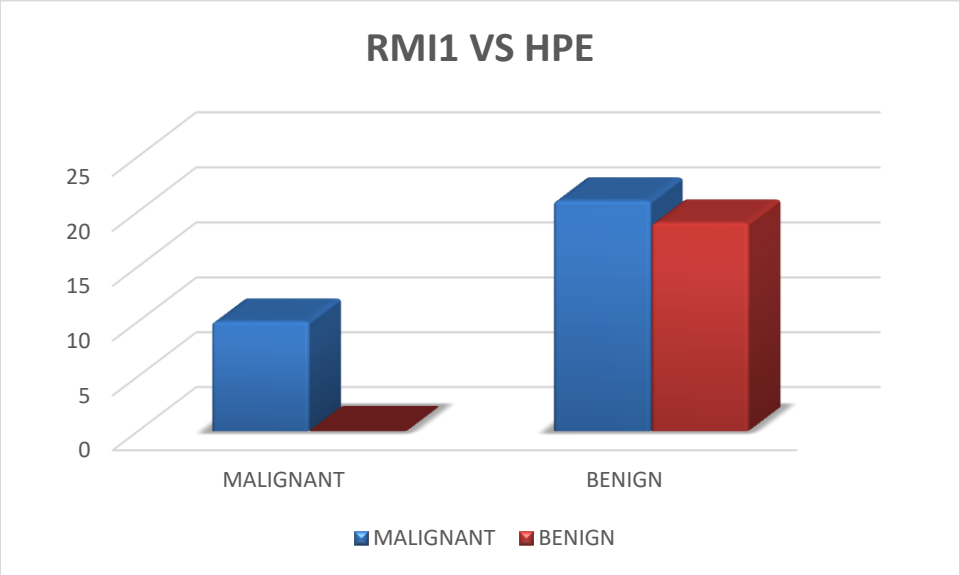
RISK OF MALINANCY INDEX

RMI 1

RMI 1	NO OF PATIENTS	PERCENTAGE
MALIGNANT	31	62%
BENIGN	19	38%



RMI 1 VS HPE		
RMI 1	MALIGNANT	BENIGN
MALIGNANT	10	21
BENIGN	0	19

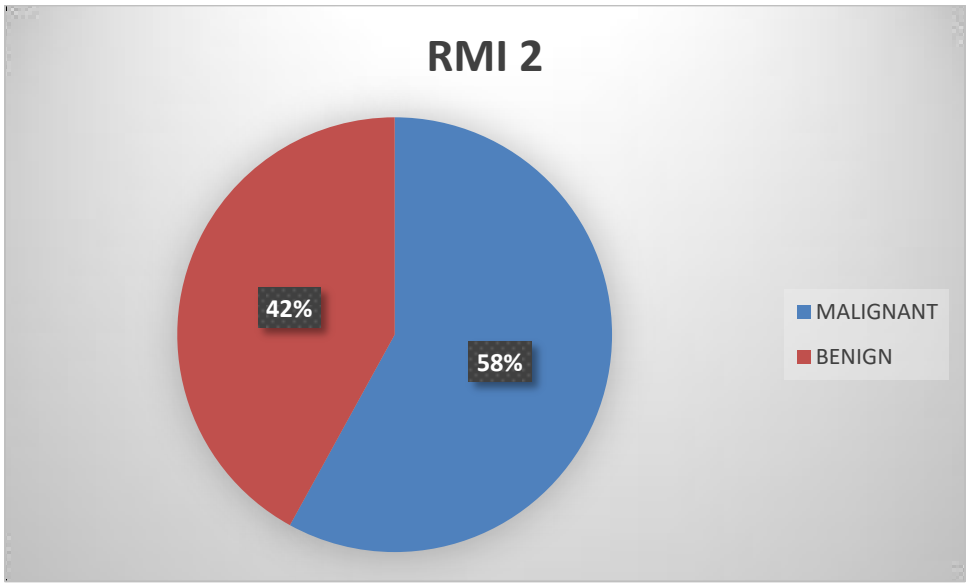


RMI 1 RESULT	
SENSITIVITY	100%
SPECIFICITY	47.50%
POSITIVE PREDICTIVE VALUE	32.26%
NEGATIVE PREDICTVE VALUE	100%
ACCURACY	58%

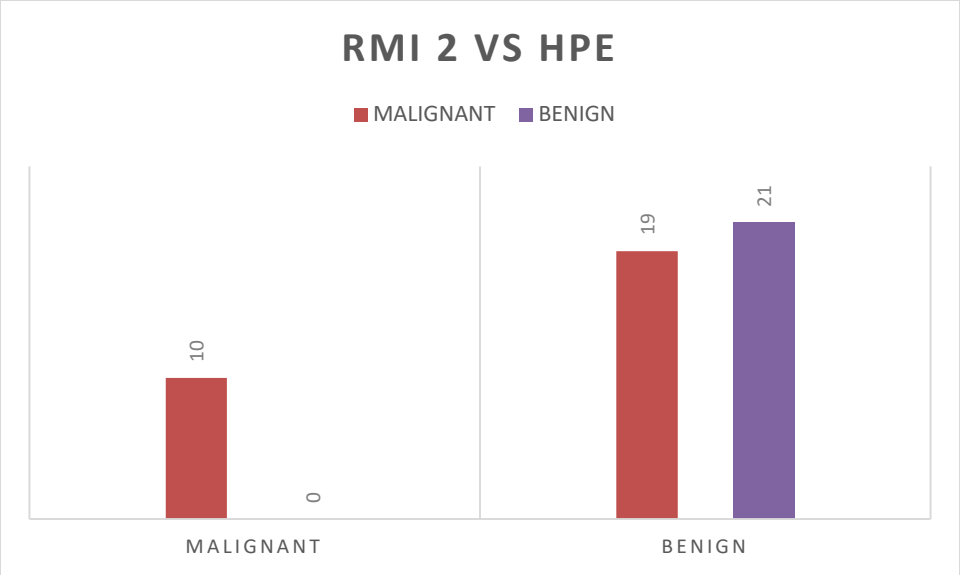
Based on scoring by RMI 1, 62% had malignant ovarian tumors, 38% had benign ovarian tumors. After RMI 1 scoring results are compared with histopathological reports only 10 patients had positive correlation with histopathological reports. In 21 patients histopathological report comes as benign but RMI 1 scoring it is belonging to malignant ovarian tumors. RMI 1 scoring system has sensitivity of 100%, specificity of 47.5%, accuracy of 58 % to differentiate benign and malignant ovarian tumors.

RMI 2

RMI 2	NO OF PATIENTS	PERCENTAGE
MALIGNANT	29	58%
BENIGN	21	42%



RMI 2 VS HPE		
RMI 2	MALIGNANT	BENIGN
MALIGNANT	10	19
BENIGN	0	21



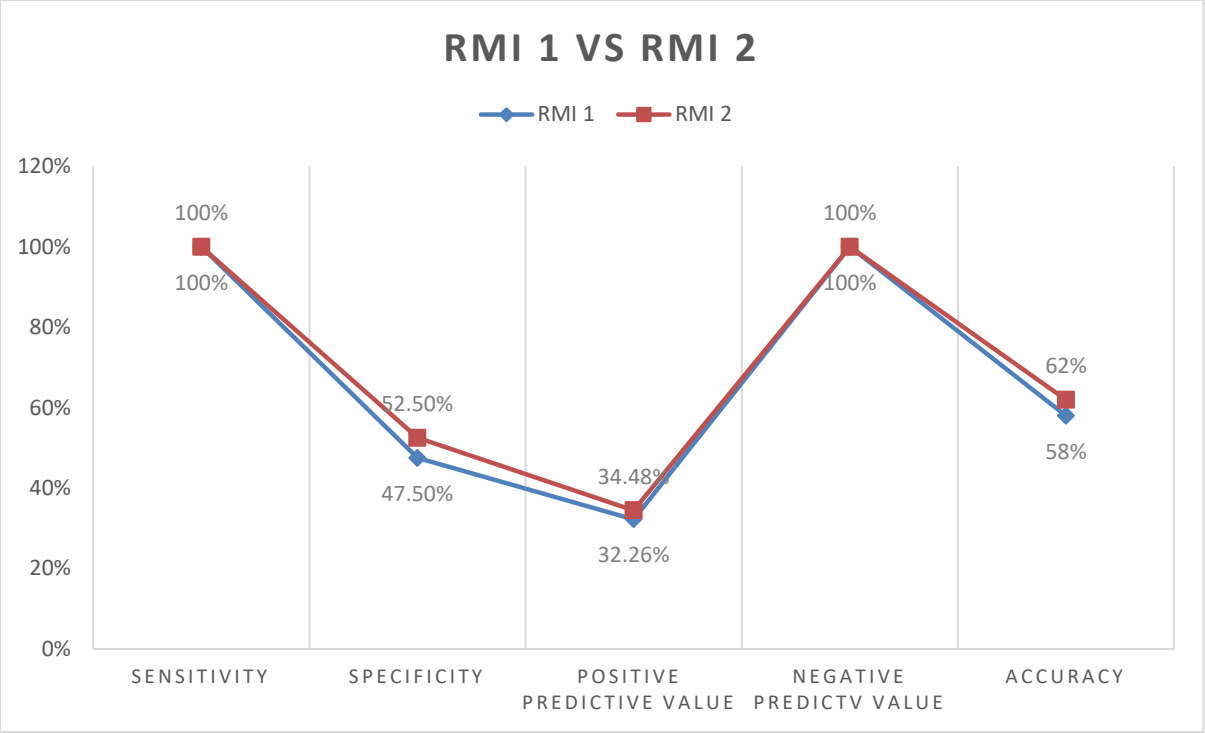
RMI 2 RESULT	
SENSITIVITY	100%
SPECIFICITY	52.50%
POSITIVE PREDICTIVE VALUE	34.48%
NEGATIVE PREDICTV VALUE	100%
ACCURACY	62%

Based on scoring by RMI 2,58% had malignant ovarian tumors, 42% had benign ovarian tumors. After RMI 2 scoring results are compared with histopathological reports only 10 patients had positive correlation with histopathological reports. In 19 patients histopathological report comes as benign but RMI 2 scoring it is belonging to malignant

ovarian tumors. RMI 2 scoring system has sensitivity of 100%, specificity of 52.5% , accuracy of 62 % to differentiate benign and malignant ovarian tumors.

COMPARISON OF RMI 1 AND RMI 2

	RMI 1	RMI 2
SENSITIVITY	100%	100%
SPECIFICITY	47.50%	52.50%
POSITIVE PREDICTIVE VALUE	32.26%	34.48%
NEGATIVE PREDICTV VALUE	100%	100%
ACCURACY	58%	62%



Based on correlation between RMI 1 and RMI 2 to differentiate between benign and malignant ovarian tumors, RMI 2 is better than RMI 1 to detect malignant ovarian tumors in the specificity of 52.5% and accuracy of 62%.

DISCUSSION

RMI is a simple scoring system, its application to evaluate ovarian tumors recommended in clinical practice. In my study cut off for RMI one and two was 200.

Ulusoy et al reported that an RMI cut off 153, there was increased incidence of malignant ovarian tumors detected.

90% of ovarian tumors diagnosed were found to be benign tumor and rest of the tumors was malignant ovarian tumor. Abdominal pain and abdominal distension was the common presenting symptoms, a significant proportion of patients were asymptomatic at the time of presentation.

Malignant ovarian tumor prevalent in women with age more than 50 years. Ascites is the most important clinical sign which predicts nature of the ovarian tumor. Patients with metastases had poor prognosis.

Tumor marker CA 125, at cut off level 35u/ml does not differentiate benign and malignant ovarian tumors. RMI scoring requires the additional use of ultrasound features. RMI 2 has the advantage over malignant tumor to distinguish between benign and malignant tumor.

DIFFERENCE BETWEEN RMI 1 & RMI 2

Feature	RMI 1	RMI 2
Ultrasound features - Multilocular cyst - Solid areas - Bilateral lesions - Ascites - Intra abdominal metastasis	0- None 1 – one abnormality 3 – two or more abnormality	0-none 1 – one abnormality 4 – two or more abnormality
Premenopausal	1	1
Postmenopausal	3	4
CA 125	U/ml	U/ml

RMI score calculated by using this formula:

Ultrasound score * menopausal score* ca 125 level (u/ml)

CONCLUSION

From this study, the following conclusions have been arrived. Among the various factors as mentioned which influence the incidence of ovarian tumors, seven factors only significantly related to malignant ovarian tumors.

1. Out of 27 patients with age >50 years, 10 patients had malignant ovarian tumors.
2. Out of 29 postmenopausal patients, 10 patients had malignant ovarian tumors. None of the premenopausal women had malignant ovarian tumors.
3. Out of 18 patients with solid areas in ultrasonography, 10 patients had malignant ovarian tumors.
4. Out of 50 patients, 19 patients had ascites in which 8 patients had histopathological report positive for malignancy.
5. Out of 50 patients, 10 patients had metastasis for them histopathological report comes as malignant ovarian tumor.
6. Out of 14 patients with abdominal pain and abdominal distension, 9 patients had benign ovarian tumors, 5 patients had malignant ovarian tumors.
7. RMI 2 is better than RMI 1 to differentiate benign and malignant ovarian tumors with sensitivity of 100%, specificity of 52.5%, positive predictive value of 34.48%, negative predictive value of 100%, accuracy of 62%.

SUMMARY

RMI scoring system is a simplest and cheapest method to differentiate benign and malignant ovarian tumor. Based on this scoring system patients refereed to gynaecological oncologist for expert management. This scoring system helps us in determining need for surgery. Appropriate care can be given for patients with gynaecological cancers. Usually treatment plans are based on the type of tumor, its stage. If ovarian cancer treated before the cancer has sprerad survival rate is 92%. Only 15% ovarian cancers are found early stage.

In my study RMI 2 scoring system has advantage over RMI 1 to identify benign and malignant ovarian tumor. Preoperative evaluation by using this scoring system avoids unnecessary surgical intervention.

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PROFORMA

Name:

Age:

Parity:

Menopausal status:

Chief complaints:

Family history:

h/o ocp’s intake:

usg findings:

CA 125 level:

CONSENT FORM

ஒப்புதல்படிவம்

பெயர் :

வயது:

பாலினம்:

முகவரி:

கோவை அரசுமருத்துவக்கல்லூரி மருத்துவமனையில் மருத்துவர் S. சுமதி தலைமையில் நடைபெறும் இந்த ஆய்வில் முழுசம்மதத்துடன் கலந்துகொள்ளசம்மதிக்கிறேன். இந்த ஆய்வில் என்னை பற்றி விவரங்களை பாதுகாப்புடன் இந்த ஆய்வில் வெளியிடஆட்சேபணை இல்லை என்று தெரிவித்துக்கொள்கிறேன்.எந்த நேரத்திலும் ஆய்வில் இருந்து எந்த நேரத்திலும் விலக்கிக்கொள்ளும் உரிமை உண்டு என்று அறிவேன்.

இடம்:

தேதி:

கைகெயாப்பம்/ரேகை

MASTER CHART

NAME	AGE	PARITY	MENOPAUSAL STATUS	CA 125	USG FINDINGS -SITE/LOCULARITY/SOLID AREAS/ASCITES/METASTASIS					SYMPTOMS	OCP	FAMILY HISTORY	RMI 1	RMI 2	HPE
RANI	65	P2L2A1	POSTMENOPAUSAL	45	U/L	MULTILOCULAR	SOLID AREAS	_	_	ABDOMINAL MASS	NO	NIL	151.2	180	BENIGN
CHINNAPONNU	42	P3L3	PREMENOPAUSAL	47	B/L	MULTILOCULAR	_	_	_	ABDOMINAL PAIN	NO	NIL	195	180	BENIGN
AANDAMMAL	65	P4L4	POSTMENOPAUSAL	9.1	U/L	MULTILOCULAR	_	_	_	ABDOMINAL DISTENSION	YES	NIL	27.3	36.4	BENIGN
RUKMANI	44	P2L2	PREMENOPAUSAL	23	B/L	MULTILOCULAR	_	_	_	ABDOMINAL MASS	NO	NIL	69	92	BENIGN
POONGODI	45	P2L2A1	PREMENOPAUSAL	31	U/L	UNILOCULAR	_	_	_	ASYMPTOMATIC	NO	NIL	0	0	BENIGN
ROSSY	60	P6L6	POSTMENOPAUSAL	132	U/L	MULTILOCULAR	_	ASCITES	_	ABDOMINAL DISTENSION	YES	NIL	1188	2112	BENIGN
THULASIYAMMAL	50	P4L4	PREMENOPAUSAL	27.1	B/L	MULTILOCULAR	_	_	_	ASYMPTOMATIC	NO	NIL	81.3	108.4	BENIGN
KALEESHWARI	47	P3L2	PREMENOPAUSAL	89	U/L	UNILOCULAR	_	_	_	ASYMPTOMATIC	NO	NIL	0	0	BENIGN
DEVI	42	P2L2	PREMENOPAUSAL	32	B/L	MULTILOCULAR	_	_	_	ABDOMINAL PAIN	NO	NIL	32	32	BENIGN
HABIBUN NISHA	46	P3L3	PREMENOPAUSAL	28	B/L	UNILOCULAR	_	_	_	ASYMPTOMATIC	YES	NIL	28	28	BENIGN
PADMAVATHY	73	P5L5	POSTMENOPAUSAL	717	U/L	MULTILOCULAR	SOLID AREAS	ASCITES	INTRAABDOMINAL METASTASIS	ABDOMINAL DISTENSION	NO	NIL	6493	11472	MALIGNA
NAGAMMAL	45	P4L2	PREMENOPAUSAL	35	U/L	MULTILOCULAR	_	_	_	ASYMPTOMATIC	NO	NIL	35	35	BENIGN
MAYILAMMAL	55	P3L3	POSTMENOPAUSAL	149.3	U/L	MULTILOCULAR	SOLID AREAS	ASCITES	_	ABDOMINAL DISTENSION	NO	NIL	1343	2388	BENIGN
VEERAMMAL	50	P2L2	POSTMENOPAUSAL	92	U/L	MULTILOCULAR	SOLID AREAS	_	_	ASYMPTOMATIC	YES	NIL	828	1472	BENIGN
PARVATHY	52	P2L2A1	POSTMENOPAUSAL	112	B/L	MULTILOCULAR	SOLID AREAS	ASCITES	_	ABDOMINAL MASS	NO	NIL	1008	1792	BENIGN
SATHYABAMA	65	P6L4	POSTMENOPAUSAL	512	B/L	MULTILOCULAR	SOLID AREAS	ASCITES	_	ABDOMINAL MASS	NO	NIL	4608	8192	BENIGN
NAGARANI	64	P2L2A2	POSTMENOPAUSAL	448	B/L	MULTILOCULAR	SOLID AREAS	ASCITES	_	ABDOMINAL PAIN	YES	NIL	4032	7168	BENIGN
NAGAMANI	45	P2L2	PREMENOPAUSAL	123	B/L	UNILOCULAR	_	_	_	ASYMPTOMATIC	YES	NIL	123	123	BENIGN
PALANAL	60	P3L2A1	POSTMENOPAUSAL	142	B/L	MULTILOCULAR	_	ASCITES	_	ABDOMINAL DISTENSION	NO	NIL	1278	2272	BENIGN
NAGAMANI	50	P5L3A2	PREMENOPAUSAL	45	B/L	UNILOCULAR	_	_	_	ABDOMINAL DISTENSION	NO	YES	45	180	BENIGN
PALANIYAMMAL	67	P2L2	POSTMENOPAUSAL	612	U/L	MULTILOCULAR	SOLID AREAS	_	_	ASYMPTOMATIC	NO	NIL	5508	9792	BENIGN
ANGATHAL	49	P3L3	PREMENOPAUSAL	170	U/L	MULTILOCULAR	_	_	_	ASYMPTOMATIC	NO	NIL	170	170	BENIGN
RANGATHAL	62	P4L4A1	POSTMENOPAUSAL	763	U/L	MULTILOCULAR	SOLID AREAS	_	INTRAABDOMINAL METASTASIS	ABDOMINAL PAIN	NO	NIL	6867	12603	MALIGNA
GOVINDHAMMAL	50	P7L6	PREMENOPAUSAL	88	B/L	MULTILOCULAR	_	_	_	ABDOMINAL DISTENSION	NO	NIL	264	352	BENIGN
AMMAPILLAI	51	P3L3A1	POSTMENOPAUSAL	678	B/L	MULTILOCULAR	SOLID AREAS	ASCITES	INTRAABDOMINAL METASTASIS	ABDOMINAL DISTENSION	NO	NIL	6102	10848	MALIGNA
CHELLAKUTTY	43	P2L2	PREMENOPAUSAL	102	B/L	UNILOCULAR	_	_	_	ASYMPTOMATIC	YES	NIL	102	102	BENIGN
MALLIKA	69	P2L2	POSTMENOPAUSAL	352	B/L	MULTILOCULAR	SOLID AREAS	ASCITES	INTRAABDOMINAL METASTASIS	PAIN ABDOMEN	NO	YES	3168	5632	MALIGNA
RANI	62	P3L3	POSTMENOPAUSAL	355	U/L	MULTILOCULAR	_	ASCITES	_	ABDOMINAL PAIN	NO	NIL	3195	5680	BENIGN
PREMAVATHY	47	P3L2	PREMENOPAUSAL	75	B/L	UNILOCULAR	_	_	_	ASYMPTOMATIC	YES	NIL	75	75	BENIGN
VASANTHAKUMARI	52	P2L2	POSTMENOPAUSAL	606	B/L	MULTILOCULAR	SOLID AREAS	ASCITES	INTRAABDOMINAL METASTASIS	ABDOMINAL DISTENSION	NO	NIL	5454	9696	MALIGNA
AMSAVENI	61	P3L3	POSTMENOPAUSAL	268	B/L	MULTILOCULAR	_	ASCITES	_	ASYMPTOMATIC	NO	NIL	2412	4288	BENIGN
ALAMELU	55	P2L2	POSTMENOPAUSAL	889	U/L	MULTILOCULAR	SOLID AREAS	ASCITES	INTRAABDOMINAL METASTASIS	PAIN ABDOMEN	NO	NIL	8001	14224	MALIGNA
BHUVANESHWARI	49	P2L2A1	PREMENOPAUSAL	99	B/L	UNILOCULAR	_	_	_	ASYMPTOMATIC	NO	NIL	99	99	BENIGN
CHITHRA	42	P2L2	PREMENOPAUSAL	146	U/L	MULTILOCULAR	_	_	_	ASYMPTOMATIC	YES	NIL	146	146	BENIGN
MONIKAMARY	53	A1	POSTMENOPAUSAL	1088	B/L	MULTILOCULAR	SOLID AREAS	ASCITES	INTRAABDOMINAL METASTASIS	ABDOMINAL DISTENSION	NO	YES	9792	17408	MALIGNA
SUSILA	60	NULLIGRAVIDA	POSTMENOPAUSAL	365	U/L	MULTILOCULAR	_	_	_	PAIN ABDOMEN	NO	NIL	1095	1460	BENIGN
LATHA	48	P2L2	PREMENOPAUSAL	189	B/L	UNILOCULAR	_	_	_	ABDOMINAL PAIN	NO	NIL	189	189	BENIGN
SENBAGAVALLI	63	P4L4	POSTMENOPAUSAL	560	U/L	MULTILOCULAR	SOLID AREAS	_	INTRAABDOMINAL METASTASIS	ABDOMINAL DISTENSION	NO	NIL	5040	8960	MALIGNA
LAKSHMI	54	P2L2A1	POSTMENOPAUSAL	309	B/L	UNILOCULAR	SOLID AREAS	ASCITES	_	ABDOMINAL DISTENSION	NO	NIL	2781	4944	BENIGN
PUSHPA	48	P5L3	PREMENOPAUSAL	108	U/L	MULTILOCULAR	_	_	_	ASYMPTOMATIC	NO	NIL	108	108	BENIGN
SHANTHI	59	P2L2	POSTMENOPAUSAL	654	U/L	MULTILOCULAR	SOLID AREAS	ASCITES	INTRAABDOMINAL METASTASIS	PAIN ABDOMEN	NO	NIL	5886	10464	MALIGNA
RAJESHWARI	61	P3L3	POSTMENOPAUSAL	439	U/L	MULTILOCULAR	_	ASCITES	_	PAIN ABDOMEN	NO	NIL	3951	7024	BENIGN
DHANALAKSHMI	57	P3L2	POSTMENOPAUSAL	678	U/L	MULTILOCULAR	_	ASCITES	_	ABDOMINAL PAIN	NO	NIL	6102	10848	BENIGN
KALYANI	48	P2L1	PREMENOPAUSAL	134	U/L	MULTILOCULAR	_	_	_	ASYMPTOMATIC	NO	NIL	134	134	BENIGN
CHELLAMAL	56	P3L3	POSTMENOPAUSAL	389	B/L	MULTILOCULAR	_	_	_	ABDOMINAL DISTENSION	NO	NIL	3501	6224	BENIGN

VALLI	66	P4L4	POSTMENOPAUSAL	998	B/L	MULTILOCULAR	SOLID AREAS	ASCITES	INTRAABDOMINAL METASTASIS	ABDOMINAL PAIN	NO	NIL	8982	15968	MALIGNA
KUPPAYI	49	P3L2A1	PREMENOPAUSAL	332	U/L	MULTILOCULAR	_	_	_	PAIN ABDOMEN	NO	NIL	332	332	BENIGN
KANNIYAMMAL	54	P1L1	POSTMENOPAUSAL	271	U/L	MULTILOCULAR	_	_	_	ASYMPTOMATIC	NO	NIL	813	1084	BENIGN
PADMAVATHY	48	P2L2	PREMENOPAUSAL	74	U/L	MULTILOCULAR	_	_	_	ASYMPTOMATIC	NO	NIL	74	74	BENIGN
VALARMATHI	60	P3L3	POSTMENOPAUSAL	289	B/L	MULTILOCULAR	SOLID AREAS	_	_	ABDOMINAL DISTENSION	NO	YES	2601	4624	BENIGN